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VOLUME 46 SEPTEMBER 1947 NUMBER 3

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THE EXPERIENCE OF DIZZINESS

Psychosomatic Observations on Some Vestibular Types

RICHARD D. LOEWENBERG, M.D.

BAKERSFIELD, CALIF.

> Plato was right to say that good doctors themselves ought to have had the disease they want to cure and being subject themselves to the misfortunes and circumstances which they wish to diagnose. Let them catch the pox if they want to know how to cure it. I would trust such a doctor.—*Montaigne.*

PSYCHOSOMATICS IN OTOLOGY

EVERY physician and psychiatrist working in a general hospital is faced daily with the universal symptom of dizziness. Although other symptoms, like pain and anxiety, have been much investigated in their inner aspects, there exists much less interest in the experience of dizziness. The literature and the discussions among physicians hardly consider the possibility of its being closely related to fear and to physical and emotional insecurity. A recent symposium on this subject in Chicago had no neuropsychiatrist as participant.

In the following notes an attempt will be made to give to otologists the psychosomatic evaluation of this syndrome, as a supplement to the recent excellent objective investigations in this field (Atkinson [1]; Weiss [2]; Williams [3]; Brunner [4]).

Contrary to the general belief, a deeper understanding of the psychosomatic approach is not easily acquired: "The change from 'body' and 'mind' to 'body-mind' is similar to, and as far reaching as that made by the physicists in going from 'space' and 'time' to 'space-time'" (Paul [5]),

From the Neuropsychiatric Service of the Kern General Hospital (Dr. N. Rilcoff, director).

1. Atkinson, M.: Observations on the Etiology and Treatment of Ménière's Syndrome, J. A. M. A. **116**:1753 (April 19) 1941; Ménière's Syndrome and Migraine, Merck Report, July 1944; The Dizzy Patient, Eye, Ear, Nose & Throat Monthly **22**:53 (Feb.) 1943; Ménière's Syndrome: The Basic Fault, Arch. Otolaryng. **44**:385 (Oct.) 1946.

2. Weiss, S.: The Pharmacopeia and the Physician; The Treatment of Vertigo and Syncope, J. A. M. A. **118**:529 (Feb. 14) 1942.

3. Williams, H. L.: The Syndrome of Physical or Intrinsic Allergy of the Head, Proc. Staff Meet., Mayo Clin. **20**:373 (June 13) 1945.

4. Brunner, H.: Ménière's Syndrome, Laryngoscope **49**:877 (Oct.) 1939; Present Status of Ménière's Syndrome, Arch. Otolaryng. **44**:38 (July) 1946.

5. Paul, L.: Implications of General Semantics for Psychosomatic Medicine, Psychosom. Med. **7**:246 (July) 1945.

and, to add Weiss's [6] pertinent statement, "it does not mean to study the soma less; it only means the psyche more."

Here I shall try a different approach to the problem than is usually chosen. The self observation of the sensations and associated emotions experienced during different kinds of vestibular attacks might lead to a better understanding of the underlying dynamic factors. Sensations and emotions are at the same time physiologic and psychologic. Physicians observe them in their daily clinical experience. This approach is not popular these days. It is the established opinion that "the possibilities of research by simple means have largely disappeared. Expensive apparatus, great amounts of expendable materials and proper assistance, secretarial and technical, are essential in modern research."[7] In a comment relating to a review of Karen Horney's book "Our Inner Conflicts" published in *The Journal of the American Medical Association* [8] an anonymous writer assures us that "the descriptive, impressionistic approach to medicopsychologic problems has seen its day." Fortunately, in the history of medicine death sentences are not quite as definite and fatal as in life. If the interest in and understanding of psychosomatics is more than a passing fashion, it is difficult to see the justification for such categoric condemnations. It is not a matter of mind or body, structural or functional, but of reversible and irreversible, as the late Smith Ely Jeliffe stressed for decades. There is always a danger of overrating an obvious symptom as cause or motive in somatic or psychologic medicine. Psychosomatic relationships are not superficial and simple but are anchored in the misty depth of the past.

Dizziness as a syndrome may well serve to illustrate the problem for the otologist. The often regretted inadequacy of language makes it imposssible to differentiate the many kinds and degrees of what is called, in a sweeping way, dizziness or vertigo. A drink makes one dizzy. Altitude makes one dizzy. A crowd makes one dizzy. The sea makes one dizzy. "Falling" in love makes one dizzy. It seems one may become dizzy at all sudden, unexpected incidents. Do all these expressions signify the same sensation and experience, differing only in degree, or are they perhaps different in kind? These questions are of much greater practical value than is usually realized and cannot be contemptuously discarded as "painful elaboration of the obvious."

Flight surgeons were fully aware of the intricate relationship of fright and motion sickness, irrespective of the most careful previous

6. Weiss, E., in Galdston, I., and others: Modern Attitude in Psychiatry: The March of Medicine, 1945, New York, Columbia University Press, 1946.

7. Cannon, W. B.: The Way of an Investigator: A Scientist's Experiences in Medical Research, New York, W. W. Norton & Company, Inc., 1945.

8. Horney, K.: Our Inner Conflicts, reviewed, J. A. M. A. **130**:247 (Jan. 26) 1946.

screening. Otologists need only to be reminded of the many insurance cases in which the complainant speaks of dizziness following injuries of the head (Ruesch and Bowman [9]). Although tests are most desirable and helpful, I feel that a good description of dizziness helps toward a preliminary, tentative localization. It is quite important to know whether the patient suffers from acute attacks of rotary vertigo, from spells precipitated by bending and turning of the head or from feelings of being drunk.

By no means is it intended to ignore, or to deny, the predominant influence of physiologic factors. It is proposed here to concentrate on the associated psychologic experiences, resisting the temptation to lose oneself in interpretations and speculations based on the modern theories of the subjective experience of time and space in contrast to the objective time and space of physics. Our present purpose is much more modest: to add to the objective findings reports of the inner experiences, which are also clinical facts. This may or may not offer material for others' interpretation, since that cannot be the present task. He who experiences something should not deceive himself that this fact alone gives him the explanatory clue. Being himself concerned, he cannot ignore his own blindspot. He may be justified in assuming that innumerable others have undergone the same experiences of physiologic events without trying to describe them. I do not think for a moment that my own experiences are unique in any way. I am convinced of the contrary. This conviction is my justification—so well formulated by Trigant Burrow: "The neurologist deals with the physiology we see, while the psychopathologist deals with the physiology we feel."

The pitfalls and errors of self observations are evident enough to need no further discussion here. However, one should differentiate between the questionable value of self analysis and self knowledge, on one side, and the value of trained self observation, which has been considered legitimate not only by artists and writers who produced self portraitures, but also by many physicians and exact natural scientists. This is sometimes forgotten nowadays. Wertham's [10] recent report of his thrombophlebitis and operation stimulated new interest in, and reassumption of, an old tradition of the history of medicine.

Self observations proved to me that there are many different types of dizziness of which a patient might be well aware and that, contrary to frequent opinions, dizziness is not necessarily a mild degree of fainting. The auras of some persons who are epileptic, moreover, may suggest

9. Ruesch, J., and Bowman, K. M.: Prolonged Post-Traumatic Syndromes Following Head Injury, Am. J. Psychiat. **102**:145 (Sept.) 1945.

10. Wertham, F.: A Psychosomatic Study of Myself, J. Clin. Psychopath. **7**:373 (Oct.) 1945.

this generalization. The usual sweeping and brief statements in the literature that concentrates on the physiologic theories entirely neglect the patient's insistence on his own experiences. "The patient is always right." A finer analysis of symptomatology has often been the forerunner of therapeutic progress. One needs only to be reminded of the history of brain tumors, which were symptomatically worked out for decades before the hopeless therapeutic outlook cleared up.

The problem of losing equilibrium has been especially well defined by Schilder,[11] who called the vestibular apparatus "the unifying factory among the senses."

> Dizziness is a psychological sign of a sensual or moral disorientation in the world . . . a danger signal as important as anxiety. It is the first sign that we are no longer sure what to do . . . in the beginning of almost any organic disease . . . [and] . . . in almost every neurosis.

The following notes were written down at the insistence of colleagues who regretted the lack of self observations. The clinical facts of the case history are given: "I do not teach, I report" (Montaigne).

REPORT OF A CASE

The patient's maternal grandmother and mother suffered from migraine. Six years ago a brother of the patient, two years older, had attacks of dizziness which cleared up after several months. The patient, 47 years old, had suffered since the age of 20 from early morning attacks of migraine, occurring at the rate of about one a month, which are described in subsequent paragraphs. There was no history of any aural condition at any time.

In 1933 the patient underwent appendectomy. In 1935 a felon of the right thumb was incised; tetanus antitoxin was given at that time. The patient had been healthy and active in his profession, working vigorously for the last decade, without vacations. On Nov. 20, 1945 he received a deep puncture wound and, having forgotten the earlier injection, another injection of tetanus antitoxin. On November 28, at 10:50 p. m., he was called to the hospital for consultation. He was feeling perfectly well when he arrived, but on entering a freshly painted ward, suddenly he could not stand up because objects of the outside world started to rotate to the right. With difficulty he finished the examination, felt somewhat improved when lying down, but did not dare to drive his car home. At no time was there any loss of consciousness, nor was there ringing in the ears. The next morning he started his office work as usual, but at 9:45 a. m. another attack occurred. The outside world moved to the right as if he were on a merry-go-round. This occurred as suddenly as the turning on of an electric switch. It was followed by nausea and violent vomiting, but no trace of headache. Reading was no longer possible. A prescription was still written, with the greatest effort of concentration. There was a strong tendency to close the eyes. Orientation was well preserved, and all questions were promptly answered in the unwarranted, somewhat silly joking vein that Wertham[10] described so

11. Schilder, P.: Psychotherapy, New York, W. W. Norton & Company, Inc., 1938, p. 44.

pointedly as "whistling in the dark." The patient was hospitalized immediately; observations, findings and laboratory tests were as follows:

Physical and Laboratory Findings (from the hospital record).—The blood pressure was 170 systolic and 110 diastolic. The pupils were equal and reacted to light and accommodation; there were no fundus changes. The ears, the nose and the throat were grossly normal. There was rigidity of the neck. The lungs were clear. The heart action was regular, with first sound muffled; there was slight enlargement to percussion. The abdomen revealed no tenderness. The extremities showed no edema. The reflexes were hyperactive and equal; there were no pyramidal signs and no motor weakness. Over both elbows and knees small multiple petechiae were noted. The sensorium was "apparently normal"; there were "no mental symptoms." The urinalysis gave essentially negative results. The red blood cell count was 4,600,000; the white blood cell count, 12,600; the hemoglobin values were 92 per cent and 14.4 Gm. The differential leukocyte count was: stab forms 3, segmented forms 84 and lymphocytes 13 per cent; there was slight toxic budding of the nuclei of polymorphonuclear leukocytes. The Kahn test was negative. The blood nonprotein nitrogen was 46 mg. per hundred cubic centimeters. The electrocardiogram showed left axis deviation.

Treatment.—A high calory liquid diet, pentobarbital sodium, phenobarbital sodium, nicotinic acid, thiamine hydrochloride and ammonium chloride were prescribed.

Course in Hospital.—The patient was insensitive to histamine. Cold water irrigation of both ears did not increase nystagmus or produce nausea or vomiting. There was slow improvement, the patient being discharged December 4.

Final Diagnosis.—The diagnosis was probable serous labyrinthitis on the basis of serum sickness.

Course After Discharge from Hospital.—During the following weeks the patient felt well when lying still on the left side and as long as the head was not moved. There was no awareness of nystagmus but there was a certain pulling, as with rubber bands, when the eyes were in end position. During the first days there was little awareness of the passing of time, partly owing to sleepiness, partly to indifference. At night the patient would awake because of dizziness when the head was turned to the right side. Certain ideas returned to him over and over again, like mild obsessions—for instance, a long forgotten verse which was the topic of a high school composition, running in translation like this:

> "Between the rim of the goblet and your lip,
> Hovers the darker powers' grip."

When he was again able to read, it seemed to him—out of proportion to any likelihood—that he had read the same texts before. (*Deja vu* syndrome?) For the next weeks any sudden turn of the head reminded him immediately that he was not quite well. There was still a "hangover" feeling which only very gradually disappeared during the following months, long after work had been resumed. Driving a car still caused some discomfort when backing up or sharp turns were made. At times there were slight spells of tinnitus; however, there was no noticeable reduction of hearing in everyday life. The audiometric test showed no greater reduction of hearing than would be common for the age.

Although the patient was seen by a number of competent specialists, the final diagnosis was never definitely settled. There was no doubt as to the definite organic nature. The acute labyrinthitis was attributed by some to an allergic reaction to the injected tetanus antitoxin, for

which the abundant literature on after-effects of tetanus antitoxin gives only few parallels (Rowe [12]; Urbach and Wilder [13]). Others thought of acute infection of the labyrinth, possibly an abortive type of encephalitis which is endemic in this county. Finally, the temporary hypertension and the possibility of a migraine equivalent were considered, in the sense of Atkinson's definition: "Migraine is Ménière's syndrome in the brain, and Ménière's syndrome is migraine in the ear." [14] Until now there has been no recurrence. It is by no means certain that the different objective explanations rule out each other. Their discussion is of no interest here and beyond my competence.

The sensation of the objective rotary vertigo, which appeared as if switched on, and disappeared as suddenly, totally different from the vague constant feeling of dizziness, seemed especially remarkable because little attention is paid to it in the literature. As the same patient had had sufficient observations on spells of migraine and on car, sea and air sickness, the differences as he experienced them might be mentioned.

Migraine wakes the patient in the early morning with an intense scintillating scotoma, followed by a boring frontal pain, nausea, diarrhea and vomiting. Sea sickness and air sickness, especially when one is flying in rough air, start with paresthesia of the finger tips and marked perspiration, followed sometimes by slight headache and nausea. In the last labyrinthine attacks, the total absence of headache, the turning on and off of the rotation of objects, seemed especially impressive. At no time was there any doubt that it was a subjective sensation and not one of the earthquakes which in slighter degree occur here also. Finally, the experience of an acute gastrointestinal upset a few months later suggested at first the onset of another labyrinthine attack. It started after midnight, following a picnic, without any headache or dizziness, but with nausea and immediate violent projectile vomiting and diarrhea. The subjective experience was, however, different enough to doubt the assumption of an equivalent. A few days later when the rest of the family came down with the same (infections?) symptoms, the observation that the sensation was different was proved to have been accurate.

Since I have made it a special point to carefully ask my patients what they mean by "dizziness," I have got a great number of confirming picturesque statements. One patient described his dizziness as "like a water box in my head swapping up and down." Another asked

12. Rowe, A. H.: Clinical Allergy in the Nervous System, J. Nerv. & Ment. Dis. **99**:834 (May) 1944.

13. Urbach, E., and Wikder, J.: Ménière's Syndrome of Allergic Origin, Med. Klin. **30**:1420 (Oct. 26) 1934.

14. In discussion on Thomas, W., and Butler, S.: Intravenous Histamine Treatment of Migraine, J. Nerv. & Ment. Dis. **103**:389 (April) 1946.

whether there was something like a cyst under his skull. Von Frankel-Hochwart [15] gave a patient's description of rotary vertigo most dramatically: "like wrapped in a towel whose ends somebody grips and then whirling around the whole." One of my patients, a hard working, ambitious lawyer from Berlin, got his first violent spell of vertigo on the day before the wedding of a casual girl friend. His neurologic and otologic work-up gave completely negative results. He described his sensation as an anxiety of slipping off to the right, as if the bed rolled around its longitudinal axis. He had never thought that his acquaintance was more than a platonic friendship. Months later, however, he volunteered spontaneously that his violent body reaction had convinced him that he was much more deeply involved than he consciously knew at the time. When he was called two years later to the Gestapo Headquarters, he had a milder recurrence of his spell.

I have observed states of chronic vertigo in many patients over many years. I recently saw a 37 year old woman whose vertiginous ailment was of nine years' standing. She had been treated as having a low grade infection and had been neurosurgically examined and operated on for abdominal complaints many times. The negative physical findings and a long history of domestic misery and of frustrations made it clear that the patient's deep depression was not a reaction to her whirling sensations but the primary cause. Mental suffering disguises itself often behind somatic symptoms. These may be a manifestation of borderline syndromes, proving the closeness of some severe neurosis to psychosis.

There are, of course, a great number of cardiac, gastrointestinal and neurologic conditions (multiple sclerosis, epilepsy, tumors and others), as well as experimental factors, that are associated with complaints of episodes of dizziness of peculiar kinds. Penfield produced dizziness through stimulation of the superior temporal convolution. In this context one has to confine oneself, however, to the aforementioned everyday types, so common in the otologist's practice, because the vestibular types of dizziness illustrate so well the contribution of a psychosomatic reaction.

THE PSYCHOPATHOLOGIC CONSTITUTION OF THE DIZZY PERSON

The inner structure of the psychosomatic relationship has been a perennial topic in the history of medicine (Loewenberg [16]) and the object of much intense recent work Although both ends of a most

15. von Frankel-Hochwart, L.: Der Ménière'sche Symptomenkomplex, ed. 2, Vienna, A. Holder, 1906, p. 42.

16. Loewenberg, R. D.: The Significance of the Obvious: Eighteenth Century Controversy on Psychosomatic Principles, Bull. Hist. Med. **10**:666 (Dec.) 1941.

intricate system are recognized and well established, the workers on the linking tunnel, started on both sides of the mountain, will still have a long time to wait until they meet. Dizziness as a syndrome claims the same challenge for the otologist as does hypertension for the cardiologist or dysmenorrhea for the gynecologist. These occurrences are often more than local symptoms; they are also responses and reactions of a personality, in which sometimes defenses of a whole lifetime become visible. Freud himself warned against hasty conclusions and once, in a discussion, considered efforts in this direction not too enthusiastically, but only with "benevolent scepticism."

The necessary clarity of methods, so aptly demanded by Solomon [17] recently, is unfortunately refuted by the nature of the matter itself, which resists sharp demarkation lines. He defines:

> The activities within the psychophysiologic organism, or body, are divided into psychologic and physiologic, both of which are bodily, or organismal activities. . . . One must carefully differentiate between mentation, or psychologic activity, and emotional activity, which is psychophysiologic.

As a clinician one is justified in relying on an empiric approach. From the patients, one can easily learn that any symptom is experienced by them as having not only a cause but also a meaning. Trained skepticism might discard such thoughts as unscientific. The patient, nevertheless, often considers his disease as a punishment. Three different approaches help the clinician along the way of such understanding: the wisdom of the language, which as in amber embeds old prerational knowledge, the study of dreams in which one reels and falls so often and the observation of infants learning how to walk.

The semantic aspect of the original usage of the English words "dizzy" and "giddy" confirm their universal psychosomatic nature, just as does that of the more common terms "pain" and "tension." They are at the same time psychologic and physiologic. The original, but still provincial, synonym of "dizzy" is "foolish." According to the great Oxford English Dictionary, the more common meanings are mentally unsteady and whirling with mad rapidity. Giddy is mostly used in the same way and originally means "possessed by a god." Giddy brain and giddy head can just as well mean a spatial as a mental condition. The close relationship between looking down and dizziness in a literary and mental meaning was quite aptly expressed by Shakespeare: ". . . How fearful, And dizzy 'tis, to cast one's eyes so low." [18] Finally, the German word for dizziness is *Schwindel,* deriving from a root meaning to fade away, to faint, and used not only for dizziness but

17. Solomon, M.: Psychophysiologic Interrelationships, Arch. Neurol. & Psychiat. **55**:678 (June) 1946.

18. King Lear 4:6:12.

also, as in English, by extension of meaning, for moral instability, and so for swindler and swindling. Its root is preserved in the little used old word "swim," meaning dizziness. The Greek word "scotoma" seems to concentrate on the blackout sensation of dizziness. In the romance languages the Latin root is visible everywhere: vertigo, vertere (to turn, as in "revert"). Even the Chinese term *Tao Yün* means bodily and mental dizziness.

That there are certain relationships between the type of personality and the disposition to react with vestibular disturbances has been suspected for a long time and has recently been made the subject of psychoanalytic studies on the neurotic character of migraine and related conditions. In some of the patients whom I have observed, I have noted some of the traits given in further detail by Fenichel.[19] He also stressed the infantile connections of the archiac sensations of equilibrium and space which people experience before they lose consciousness when they fall asleep. This experience is a revival of the early ambivalent experience of pleasure and fear. The great importance of kinesthetic impressions of infancy was pointed out by Freud many decades ago. In his interpretation of dreams he wrote . . . "The exciting games of childhood are repeated in dreams of flying, falling, reeling and the like, but the sensuous feelings are now transformed into anxiety" [20] . . . and later he mentioned the obvious symbolic meaning of the anxiety in dreams of falling in women. Fenichel [19] expressed the opinion that in the infant the fear of losing stability may form the pattern for the structure of later anxieties. The sensation that some people get of being dizzy and nauseated when they are suddenly awakened at night—for instance, by the shrill ringing of the telephone—might be a repetition of these early experiences.

Another question is why these conditions seem to be more prone to occur in men than in women. Walsh and Adson [21] reported a ratio of 136 male to 50 female patients. Shakespeare saw this long ago: "Our fancies are more giddy and infer more longing, wavering, sooner lost and worn, than women's are." [22]

One could not better express what is called in contemporary jargon the ego-closeness of these sensations, which makes them so distressing

19. Fenichel, O.: The Psychoanalytic Theory of Neuroses, New York, W. W. Norton & Company, Inc., 1945, p. 253. He enumerates these traits as typical: overconscientiousness, readiness to accept blame, parental attachment, inhibitions, etc.

20. Freud, S.: Interpretations of Dreams, in The Basic Writings of Sigmund Freud, translated and edited by A. A. Brill, New York, Modern Library, 1938, pp. 315 and 316.

21. Walsh, M. N., and Adson, A. W.: Ménière's Syndrome, J. A. M. A. **114**:130 (Jan. 13) 1940.

22. Twelfth Night 2:4:29.

to the patient. This very fact might also account for the common observation, often forgotten by physicians, that the doctor, as in this case also, cannot always make a simple diagnosis of his own illness when he is emotionally involved. Flight surgeons have reported that a recent accident in training immediately swelled the number of "washouts" among the cadets who previously proved to be stable and fearless. In my case, typical conflicts of middle age and anxieties over a pending board examination might have contributed to revive older vestibular mechanisms. With their threshold lowered, they responded, therefore, to the allergic stimulus before more typical reactions occurred.

One can approach the problem from the other side and ask not only how the personality contributed to the disease, but also how the disease influenced the personality. Goldstein stressed "this struggle of the changed organism to cope with the defect" in his studies on after-effects of brain injuries. Why is dizziness such an intense sensation, often going far beyond the tortures of anxiety or pain? Fainting, even when it does not occur, is feared and expected by the sufferer. The famous philosopher Kant characterized the preceding change of many unequal sensations beyond our comprehension as a "prelude to death." In the more detailed quoted case, it can be stated that the intellectual awareness of human frailty daily impressed on all doctors, became an emotional experience of losing ground, a threat to his own existence, when the world began to shake. This was like a reminder: "You too," especially so since at the same time a number of close friends and colleagues of his age suddenly died. When in younger years a much more dangerous operation had to be performed for acute appendicitis, the appendix just rupturing, no such reactions occurred. Moreover, rather late in life an aroused interest in classic, meditative music was further stimulated by a so far unwarranted fear of loss of hearing. Otherwise, the established pattern of behavior continued unchanged. There was no illusion about the possibility of an early recurrence with more incapacitating effects, but this was much less disturbing than expected.

Therapeutically, all the usual remedies were applied, none with any striking effect. It occurred to me, however, that perhaps one might try some of the newer mixtures of scopolamine and barbiturates for these conditions, as they have proved their value for motion sickness. In several cases, I was favorably impressed by the results, but it would be too early to draw conclusions. I found only one similar suggestion in the literature.[23] Statistics of treatments are not highly considered, because symptomatic relief pleases the patient more than the physician.

23. Holling, H. E.; McArdle, B., and Trotter, W. R.: Prevention of Seasickness by Drugs, Lancet **1**:127 (Jan. 22) 1944.

In this connection Wolff's [24] statements on migraine are most remarkable. Irrespective of the therapy, the percentage of improvements and that of failures remained unchanged during the last decades. It is characteristic of psychosomatic conditions, like asthma, dysmenorrhea and hypertension, that the most different treatments may work at times and may be completely useless at other times. Therefore, in these cases therapy alone cannot be used as a proof for, or against, a theory of pathogenesis. This should not be interpreted as nihilism. One should, however, be aware of the many other therapeutic factors.

The cooperation and the attitude of the patient are quite important. Every physician has observed post-traumatic patients who learn after injuries of the head to avoid such turns and postures as might precipitate dizzy spells. Experiments and daily observations prove that the force of distracted attention inhibits the awareness of sensations. The car driver and the air plane pilot usually remain free from the symptoms from which their passengers suffer. In certain cases some additional deeper mechanisms may be at work for this observation: Passengers want to make sure of the possibility of escape from their own excitement and from the vehicle in which they are projected. A patient of Fenichel's felt anxiety in a car when somebody else, not when he himself, was driving: "Why should I be afraid," he said, "if I can stop the car at any time."

Any psychosomatic investigation puts an additional burden and responsibility on the physician, often far beyond his patience and time. The purpose of the foregoing remarks, however, is fulfilled if new interest has been provoked in an old problem which is perhaps not quite as settled as is sometimes believed. Nowadays only hundreds of cases are considered worth while. The individual case of one's daily practice has no scientific standing. Thus one is torn between two strivings as a physician: On the one side is the will to classify and to generalize the overwhelming diversity of phenomena into orderly laws, thus enslaving nature's infinite variety into rigid pigeonholes; on the other side, a humanly much stronger trend is stimulated by the uniqueness of the single case, of which each patient is convinced. (This explains too the general popularity of human interest stories in biographies and anecdotes.) Is it a vain hope to search for general laws in individual observations? Unlike some modern psychotherapeutic schools, who confine, for good reasons, their treatments to people they like, the otologist and the neuropsychiatrist, who is first and last a physician, cannot afford this enviable selection. His professional tradition leaves him no choice. He has to help those

24. Wolff, H. G., in discussion on Thomas, W. A., and Butler, S.: Intravenous Histamine Treatment of Migraine, J. Nerv. & Ment. Dis. **103**:393 (April) 1946.

who ask for it. The strain on him day in and day out should not be underrated. It is believed that his first scientific interest in the one patient at the moment opens the door for him to the indispensable approach to the whole personality, who wants relief from him. The always new fascination of unprejudiced observation becomes thus a help for the physician himself. His infinite task was recognized centuries ago, at a time when the term "psychosomatic" was unknown and unnecessary because it was a living reality:

When the young Goethe studied law in Strassburg, Germany, in 1770, he observed that at the dinner table the medical students were the only ones who continuously debated their science, which others completely dropped after leaving the lecture halls. And he went on to give the reason for this difference that attracted him to their company; "Medicine occupies the whole man, because it occupies itself with the whole of man."

SUMMARY

Dizziness, like asthma, hypertension and dysmenorrhea, is a physiologic and psychologic reaction. The syndrome, so universal in otologic practice, is reexamined in its psychiatric or psychosomatic aspects. Vascular, aural, subcortical and cortical mechanisms can excite the vestibular apparatus through physical, bodily and psychologic stimuli. The psychosomatic interrelationship is not obvious or superficial and cannot be easily recognized. It is anchored deep in the past. It is not entirely a matter of the outside pathogenic factor to which the organ system of the body responds with reactions. Thus, the picture of the disease can be modified by the whole organism. Tissue pathology and psychopathology are inseparable, each a different aspect of an underlying disturbance of function. The practical significance of such an approach for insurance cases and aviation medicine is discussed.

Self observation of physiologic events has always contributed to the science of medicine and has been practiced by exact natural scientists. It is a legitimate enlargement of the clinical picture and reveals more regular laws of inner experience than is usually realized. It should not be confused with self analysis and the latter's pitfalls, although it may well contribute material for others' interpretation.

A self observation of serous labyrinthitis is given in detail and compared with the same patient's experiences of migraine and motion sickness. The much claimed close relationship of fainting and dizziness is questioned; that fears and frustrations are precipitating factors is illustrated from other observations.

Careful questioning of dizzy patients confirms that they experience different kinds of dizziness, although they might find it hard to verbalize their feelings. The rotary vertigo of the acute spells and the postural dizziness are distinct entities for the patient.

The semantic discussion of the etymology of the term "dizziness" in different languages confirms the belief that it is not incidentally used simultaneously to express both the physical and the mental-moral loss of equilibrium. Modern psychoanalytic theories are reviewed, which connect the early infantile kinesthetic impressions of instability with the sensations of disturbed equilibrium of later life, revived by the mental-moral vacillations arising from anxiety and frustrations.

The question whether certain personality types are predisposed toward vestibular reactions is considered.

There are all grades of functional vestibular attacks, from reversible to irreversible, which require a combined evaluation of all possible contributing factors for a better understanding and treatment.

1830 Flower Street.

SPEECH DISORDERS IN WORLD WAR II

IX. Dysphonia

WILLIAM G. PEACHER, M.D.*
SYRACUSE, N. Y.

THE MANAGEMENT of speech disorders during World War II has been outlined in several previous communications [1] from this clinic (Brooke General Hospital, May 7, 1943 to Feb. 22, 1945; McGuire General Hospital, Feb. 22, 1945 to April 1, 1946; Valley Forge General Hospital, April 1, 1946 to date). A survey of all other hospitals with speech facilities attached to the United States and British armies was also carried out. The findings demonstrated conclusively that dyslalia and dysphasia were the most common speech defects encountered and that dysphonia was only infrequently observed. The rarity of phonatory problems is further emphasized as this study includes material collected from a maxillofacial center (Valley Forge General Hospital). In this respect, complete statistics will probably never be available, for several reasons: First, the characteristics of the voice attendant on wounds of this type are not always mentioned. Second, laryngeal injuries are not uncommonly included with gunshot wounds of the neck without complete subclassification. Finally, a high percentage of wounds of the larynx are fatal.

This paper includes material on dysphonia collected from this clinic through Aug. 1, 1946. The problem of rehabilitation has been largely neglected, the patient being dispositioned to duty or discharged, depending on the individual problem present. The literature accumulated

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* Formerly Major, Medical Corps, Army of the United States.

1. Peacher, W. G.: (*a*) Speech Disorders in World War II, J. Speech Disorders **10**:155-161 (June) 1945; (*b*) Speech Disorders in World War II: II. Further Studies, J. Nerv. & Ment. Dis. **102**:165-171 (Aug.) 1945; (*c*) Speech Disorders in World War II: III. Dysarthria, J. Speech Disorders **10**:287-291 (Dec.) 1945. (*d*) Peacher, W. G., and Peacher, G. M.: Speech Disorders in World War II: IV. Dysarthria and Dyslalia; Methods of Examination, J. Nerv. & Ment. Dis. **103**:484-493 (May) 1946. (*e*) Peacher, W. G.: Speech Disorders in World War II: V. Organization of a Speech Clinic in an Army General Hospital, J. Speech Disorders **11**:233-239 (Sept.) 1946. (*f*) Speech Disorders in World War II: VI. Survey of Speech Clinics in the United States and British Armies, J. Nerv. & Ment. Dis., to be published. (*g*) Speech Disorders in World War II: VII. Treatment of Dysarthria, ibid., to be published. (*h*) Peacher, W. G., and Harris, W. E.: Speech Disorders in World War II: VIII. Stuttering, J. Speech Disorders **11**:303-308 (Dec.) 1946.

during World War II does not record any specific data on this phase of speech disorders other than three recent reports. One, by Riseman and Aagesen,[2] concerns the laryngeal neuroses. They studied 17 patients in the Army, only 1 of whom, however, acquired his neurosis while on overseas noncombat duty. An emotional conflict was found to be the etiologic background in all cases. Fifteen were returned for further useful service after being treated by routine psychotherapeutic measures, consisting primarily of suggestion (with or without narcosis) and reassurance. The second paper, by Lederer and Howard,[3] includes a discussion of the mechanism and the management of wartime laryngeal injuries observed in 43 patients at a naval installation. These patients were classified as follows, with case reports as indicated: direct trauma, 8; indirect trauma, 2; thermal trauma, 1. The remaining patients were not subdivided in this manner, but the authors mentioned that some of the injuries were not incurred in battle (six were so cited in the case histories). A brief discussion of the value of vocal rehabilitation is included. Instruction was given several hours daily, and, in addition, the patients practiced at home. The minimum period necessary to achieve a satisfactory result in the average case is set at two months. Finally, in a report from the Surgeon General's Office, by Canfield,[4] five classes of patients are mentioned as needing to be evacuated to the zone of the interior for further specialized treatment in addition to that given for the functional neuroses with otolaryngologic symptoms. One group included those having wounds of the larynx. The need of some of these men for reconstructive surgical treatment and vocal training is stressed. Case studies, statistics and additional details are, however, not included.

Numerous papers have appeared on gunshot and shell fragment wounds of the neck with laryngeal involvement (Bruggeman[5]; Debenham[6]; Despons[7]; Evans[8]; Falk[9]; Gouvea[10]; Kelly[11]; Kuchinka[12];

2. Riseman, B., and Aagesen, W. J.: Laryngeal Neurosis Incident to Military Service, Arch. Otolaryng. **43**:22-24 (Jan.) 1946.

3. Lederer, F. L., and Howard, J. C.: Wartime Laryngeal Injuries, Arch. Otolaryng. **43**:331-343 (April) 1946.

4. Canfield, N.: Otolaryngology in the European Theater of World War II, Ann. Otol., Rhin. & Laryng. **55**:108-120 (March) 1946.

5. Bruggeman, A.: War Wounds and Their Therapy, Therap. d. Gegenw. **83**:313-318 (Sept.) 1942.

6. Debenham, R. K.: War Surgery in the Middle East, Brit. M. J. **2**:223-227 (Aug. 21) 1943.

7. Despons, J.: Emergency Therapy of Wounds and Injuries of the Larynx, Rev. de laryng. **60**:733-739 (Nov.) 1939.

8. Evans, C.: Treatment of Wounds and Injuries of Larynx and Trachea, M. Press **209**:91-93 (Feb. 10) 1943.

(Footnotes continued on next page)

Lewis [13]; Lumsden [14]; Lynch [15]; Perwitzschky [16]; Rao [17]; Reeves [18]; Trutner [19]), but surgical management has been emphasized throughout. Although impairment of voice is not mentioned in every instance, it is assumed that it was present in some patients because of the nature and the extent of the injuries. It is evident from Lewis' [18] statistics that the incidence of traumatic dysphonia is low in that only approximately 1 per cent of all wounds necessitating hospitalization involved the neck. Thirteen cases are cited in his paper; phonatory problems were present in 6, and spontaneous improvement was noted in 3.

ETIOLOGIC CONSIDERATIONS

Gunshot wounds of the cervical region may cause dysphonia by one or more of the following general mechanisms: (*a*) direct trauma of the larynx, (*b*) paralysis of the vagus or of the recurrent laryngeal nerves and (*c*) indirect injury of the aforenamed structures with resulting temporary edema, hemorrhage, etc. Many variations and combinations, however, may occur that will be commented on later. In occasional patients late extrinsic scar contracture can produce dysphonia by distortion of the laryngeal mechanism.

Other patients who may be considered as war casualties are those in whom dysphonia or aphonia has developed as a conversion symptom on the basis of hysteria caused by the rigors of combat. This problem will be discussed in a later section of this paper. As poisonous gases were

9. Falk, P.: Therapy of War Injuries of the Larynx, Med. Welt **14**:81-84 (Jan. 27) 1940.

10. Gouvea, A.: Complete Left Recurrent Laryngeal Paralysis Produced by Cervical Hematoma After Deep Bullet Wound of Neck, Rev. méd. brasil. **11**:113-116 (Aug.) 1941.

11. Kelly, H. D. B.: Oto-Rhino-Laryngology in the Navy, J. Laryng. & Otol. **60**:279-291 (July) 1945.

12. Kuchinka, A.: Injuries of Cricoid Cartillage, Monatschr. f. Ohrenh. **74**:278-285 (June) 1940.

13. Lewis, R. S.: Wounds of the Neck and Larynx, Lancet **1**:781-784 (June 17) 1944.

14. Lumsden, R. B.: Wound or Injuries Due to Explosion, J. Laryng. & Otol. **57**:385-386 (Aug.) 1942; Otolaryngology in the Army, ibid. **50**:91-109 (March) 1945.

15. Lynch, M. G.: Traumatic Injuries of the Larynx, Bull. U. S. Army M. Dept. **5**:127-128 (Feb.) 1946.

16. Perwitzschky, R.: War Wounds of the Larynx, Med. Klin. **36**:669-672 (June 21) 1940.

17. Rao, B. T.: Wounds and Injuries of the Larynx, Antiseptic **37**:753-760 (Sept.) 1940.

18. Reeves, B.: A Penetrating Shell Wound of the Neck Involving the Larynx, J. Laryng. & Otol. **58**:327-330 (Aug.) 1943.

19. Trutner, V.: Wounds and Injuries of the Larynx, Arch. Otolaryng. **36**:629-631 (Nov.) 1942.

not used during this war, injuries due to them were not observed. Finally, my associates and I studied a small number of patients whose dysphonia was due to conditions comparable to those seen in ordinary clinic practice, including tumors and inflammatory processes. Other patients falling into this group, such as those suffering from hearing loss, excessive use of the voice, delayed sexual development, and diminished breath pressure as observed in such entities as pneumothorax, cardiac decompensation and postoperative pneumonectomy, were rarely examined. Phonatory problems occurring during the course of acute, temporary laryngeal disease were not included.

Dysphonia has occurred in 12 per cent of 625 patients examined by us. In 42 (or 54 per cent) of these 78 patients it resulted from wounds or exposure to enemy action. As I have been attached to the neurosurgical service throughout, traumatic dysphonia with vocal impairment has usually been associated with some neurologic lesion, e. g., hemiplegia, injury of the brachial plexus, paralysis of the ninth,

Etiologic Factors of Dysphonia

	Cases
1. Trauma	33
2. Tumor	15
3. Neuropsychiatric conflict	14
4. Inflammatory disease	9
5. Congenital anomaly	3
6. Postoperative difficulty	2
7. Chemical injury	1
8. Thermal injury	1
	78

tenth (including the recurrent laryngeal branch), eleventh and twelfth cranial nerves and quadriplegia. Patients with dysphonia alone were usually directed to the hospital specializing in the treatment of the condition suspected to be etiologic, but if dysphonia was combined with another injury, as mentioned in a foregoing paragraph, the patient was assigned to the installation designated to clarify the major injury present. The patients whose difficulty was functional were seen in consultation with the neuropsychiatric section primarily while I was stationed at Brooke General Hospital. It is also to be pointed out that no otolaryngologic center as such was organized during World War II, although men specializing in this field were assigned to most of the general hospitals. The accompanying table shows the various etiologic factors of dysphonia encountered in this series.

EXAMINATION

A complete investigation of a case of dysphonia includes:

1. The recording of a detailed history of the case.

2. Complete physical, neurologic and laboratory examinations to rule out systemic conditions. This should include audiometry if there is marked loss of

hearing with associated impairment of voice. It is further important to inspect and palpate the laryngeal region for further evidence of the extent of trauma. Roentgen examination has been of some value when there is a space-occupying lesion, e. g., carcinoma or hematoma.

3. Evaluation of psychologic and psychiatric factors. These may be primary or contributory.

4. Adequate recording of the characteristics of the voice, including the following:

(*a*) Intensity or loudness.

(*b*) Tone or quality, e. g., hoarse, husky, whispered.

(*c*) Pitch, including a comment on inflection.

5. Examination to demonstrate the frequency and incidence of defective sounds.

6. Laryngoscopy—direct or indirect.

7. Careful investigation of other speech disorders often associated with dysphonia, particularly dysarthria, dyslalia and dysphasia. Methods of examining patients for dysarthria and dyslalia have already appeared[1d]; those for dysphasia will be published later. Functional dysphonia has not infrequently been associated with stuttering. This problem also has been discussed recently.[1h]

8. Recording of the voice. This has served as a permanent record demonstrating improvement and has also been of value during rehabilitation.

9. Photographic representation of the laryngeal apparatus as indicated.

TRAUMATIC DYSPHONIA

Laryngeal paralyses, traumatic, inflammatory, degenerative or neoplastic, of central origin have rarely been observed in our clinic. Three cases only of encephalomyelitis with signs of medullary involvement, including dysphonia, have been studied. We have treated patients with cerebellar tumor, syringomyelia, etc., with vocal impairment, but these have been included in the group with dysarthria on account of the predominating character of the latter symptom.

During and following World War I various neurologic syndromes with associated phonatory problems involving the glossopharyngeal, vagus, accessory and hypoglossal nerves due to injuries of the neck were described. These included the laryngeal syndromes of Vernet, Jackson, Collet-Sicard and Tapia. Elkin and Woodhall [20] have recently discussed cases of this type in reference to World War II, and this has also been our experience. From the standpoint of frequency, injury of the recurrent laryngeal nerve has been most commonly noted, followed closely by lesions of the retroparotid space with implication of all the last four cranial nerves (syndromes of Villaret and Collet). The other combinations have only occasionally been observed.

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20. Elkin, D. C., and Woodhall, B.: Symposium on Vascular Surgery: Combined Vascular and Nerve Injuries of Warfare, Ann. Surg. **119**:411-431 (March) 1944.

Some wounds of the neck are superficial and consequently do not interfere with laryngeal function. Others terminate fatally because of trauma of the major blood vessels, the spinal cord and other vital structures. Tracheotomy has often been necessary as a lifesaving measure when the larynx has been involved. Further, owing to the many important and closely related vital structures in the cervical region, the injuries and sequelae have generally been multiple. In addition to the aforementioned neurologic combinations, they include the following different types of traumas. It is to be noted that this list does not by any means exhaust all of the possibilities that have been studied. Then, again, in most instances the majority of the patients have had multiple wounds elsewhere in the body (head, thorax, abdomen and extremities) in addition to those productive of dysphonia. The list of traumatic lesions associated with dysphonia follows:

1. Injury of the recurrent laryngeal nerve plus quadriplegia.
2. Injury of the recurrent laryngeal nerve plus paralysis of the brachial plexus.
3. Paralysis of the recurrent laryngeal nerve.
4. Damage of the vagus nerve plus comminuted fracture of the cervical portion of the spinal column without paralysis, plus laceration of the internal jugular vein and of the common carotid artery (in some, with aneurysm formation).
5. Contusion and edema of the larynx (indirect trauma), plus trauma of the brachial plexus.
6. Indirect trauma of the larynx plus paraplegia.
7. Indirect trauma of the larynx.
8. Structural damage of the larynx, including destruction of cartilage, perichronditis, adhesions, stenosis, infections, fistulas.
9. Multiple general wounds plus injury of the recurrent laryngeal nerve plus paralysis of the brachial plexus.
10. Cervical hematoma with symptoms of pressure.
11. Injury of the major vessels (laceration, aneurysm, thrombosis, embolism) with hemiparesis plus edema of the larynx.
12. Paralysis of the vagus nerve.
13. Cervical trauma plus infection plus edema of the larynx.
14. Extrinsic scar with distortion of the laryngeal cartilages.
15. Intrinsic laryngeal trauma plus paralysis of the recurrent laryngeal nerve.
16. Paralysis of the recurrent laryngeal nerve plus injury of the cervical sympathetic nerves plus quadriplegia.
17. Laryngotracheal or bronchoesophageal fistulas with direct trauma of the larynx.
18. Esophageal diverticulum with laryngeal trauma.

Shell fragments and bullets passing close to the larynx without actually damaging the intrinsic mechanism or the nerve supply often produced temporary dysphonia (usually of one to two weeks' duration),

due to congestion and edema. Wounds necessitating tracheotomy without actual permanent trauma of a vocal cord also caused vocal impairment, which persisted after the tube was removed in some cases. Patients with involvement of both cords, either intrinsic or extrinsic in nature, or paralysis of the superior laryngeal nerve have been uncommon.

Early examination of patients with paralysis of the recurrent laryngeal nerve showed the involved cord usually to be in the midline, and in the cadaveric position in the presence of vagal lesions, according to medical records obtained from theaters of operation. When examined at this station two to three months after the trauma, either the structures had returned to normal or there was evidence of abductor paralysis in injuries of the recurrent laryngeal nerve. During this process the voice had occasionally reverted to its usual characteristics, and in those cases with continued paralysis the opposite cord was frequently seen to cross the midline for approximation during phonation. Some of these patients, however, still showed changes in pitch, tone and intensity two to three months after the original injury with associated paralysis. Vocal exercises in combination with other measures to be described and depending on the individual problem present have resulted in further improvement in many cases of this type as well as in those with moderate unilateral damage of the intrinsic laryngeal structures.

THERAPY IN CASES OF TRAUMATIC DYSPHONIA

Surgical Treatment.—The surgical procedures may be divided into:

1. Immediate procedures, including débridement and tracheotomy.
2. Reconstructive procedures. The latter include:

(*a*) Dilation of laryngeal strictures.

(*b*) Elective tracheotomy in cases in which the cannula had been inserted in the vicinity of the laryngeal cartilages during the exigencies of warfare. This procedure may also be necessary prior to further surgical treatment.

(*c*) Laryngectomy may be indicated occasionally with severe avulsion injuries.

(*d*) Procedures designed to improve the condition of patients with bilateral abductor paralysis (King's, Kelly's, Hoover's and others).

(*e*) Excision of intrinsic or extrinsic scars or both.

(*f*) Plastic repair of the larynx.

(*g*) Exploration of the vagus or the recurrent laryngeal nerves: neurolysis, anastomosis, and other procedures.

Physical Therapy.—Heating and gentle massage of the involved area together with electrical stimulation (galvanic or faradic, depending on the reaction obtained) are indicated in cases of nerve injury.

Vocal Training.—These exercises are instituted as soon as the acute pathologic reaction has subsided and are subject to change depending

on the individual circumstances. Practice sessions have usually been conducted thirty minutes twice daily. They include:

1. Breathing exercises.

2. Methods to obtain relaxation (Jacobson [21]).

3. Exercises to increase the range of tone. The aspirate approach includes suitable drills which are particularly valuable for good initiation of tone. This is done simply by beginning with "H" and going directly into a vowel. Words taken separately and in poems, with enunciation starting in this same manner, contribute further ease in production. Exercises are then introduced to further strengthen the new vocal quality, including previously defective and difficult sounds.

4. Ear training technics to improve the patient's discrimination of the proper pitch level are important.

5. Esophageal speech or the fitting of an adequate mechanical larynx or both may be indicated in laryngectomized persons.

Psychologic Technics, Including Psychotherapy.—The patient must further be made aware of his vocal defects.

Recordings of the Voice.—These allow a patient to study the defects of his own phonation and enable him to imitate the correct manner of production from a standard recording or as given by the therapeutist.

Treatment of Associated Disorders of Speech, Including Dysarthria, Dyslalia and Dysphasia.—The therapy indicated for the first two conditions has already been published [18] and that for the dysphasias will be the subject of a future publication. Speech reading exercises to prevent deterioration of speech, fitting of hearing aids and other measures have also been emphasized in those cases in which there is associated eighth nerve impairment. This problem has recently been discussed.[18]

There are many methods that may be useful with this group which have not been listed in detail at this time, as they come within the province of the speech pathologist. It has been intended only to point out their existence and value in this type of case.

NEUROPSYCHIATRIC GROUP

Symptoms associated with aphonia taken together with previous personality characteristics were usually sufficient to enable one to make a clinical diagnosis of psychoneurosis on the basis of conversion hysteria. The only exceptions were those of combat or operational fatigue. The findings in these men were similar to the findings mentioned in the foregoing sections but were transient in duration (a few days to a few weeks) and usually occurred in a previously stable person who exhibited these symptoms only under extenuating battle circumstances. The most frequent history given was that of being exposed to enemy action over a

21. Jacobson, E.: Progressive Relaxation, Chicago, University of Chicago Press, 1938.

long period, or of being covered with debris from shell and mine explosions or of minor unrelated traumas. In all the patients the condition developed in this country, with the exception of 8 who saw action, 5 of whom were wounded. In all instances laryngeal examination showed normally functioning vocal cords. Many patients in the latter category were improved early by instituting the appropriate therapy behind the lines and were returned to duty. Others were evacuated to the zone of the interior for further observation and treatment. The following measures have been successfully employed in treating patients of this type.

TREATMENT OF THE NEUROPSYCHIATRIC GROUP

The first list of therapeutic procedures consists of those that have been primarily applied to patients with acute battle-produced disorders, although they are also appropriate for similar patients in the zone of the interior:

1. Deep sedation, often for two or three days.
2. Narcosynthesis (sodium amytal, pentothal sodium) for rapid abreaction and the removal of conversion symptoms and hysterical amnesias.
3. Electroshock therapy.
4. Insulin treatment in subshock doses. This has also been of value with those soldiers who show marked loss of weight.
5. Hypnosis.
6. Psychotherapy, particularly suggestion and reassurance, sometimes combined with narcosynthesis. This was of great value. The relaxation and breathing exercises recommended by Jacobson [21] were also helpful. Spontaneous recovery was occasionally noted after accidental phonation had occurred in an unguarded moment.

In addition to the foregoing measures, others have been of value. These were more highly developed after the patients had been evacuated to the United States for continued observation and treatment and included the following:

7. Group talks or lectures (usually three times a week for one month). These were designed to give the patient an understanding of the origin and nature of his symptoms. A brief introduction to the anatomy of the nervous system was often included for additional clarification.
8. Group projection (usually given one to two times weekly during the course of rehabilitation). The staff and the patients alternated in presenting short sketches dramatizing the common experiences of previous soldiers who had recovered. The responsible physician concluded the presentation with a brief explanatory comment. This plan gave the patient additional insight as to the nature of his problem and attempted to offer a solution.
9. Reconditioning. All zone of interior installations included all forms of recreational, vocational and educational diversional activities, which were extremely important during this phase of rehabilitation.
10. Treatment of associated speech disorders. Stuttering particularly would be dealt with in this group, whether of the combat fatigue or of the psychoneurotic type. Principles of therapy in this respect have already appeared.[1h]

MISCELLANEOUS GROUPS (TUMORS, INFLAMMATORY CONDITIONS, ETC.)

Cases of dysphonia resulting from the presence of a tumor or an inflammatory lesion, improper use of the voice and other abnormalities have occasionally been noted. Cases of this type will be discussed only briefly, as they are more commonly observed in civil practice. Five patients with chronic laryngitis were examined, in one of whom the condition was syphilitic. Another showed an adjacent mediastinitis extending into the larynx. The 3 men who had encephalomyelitis with dysphonia of central origin have already been mentioned. There were 3 soldiers with congenital malformations, all with associated arrest of sexual development. There was also a patient with laryngeal stenosis as a result of taking lysol in an attempt at suicide. One man showed laryngeal damage following severe burns of the face and neck with associated inhalation of fumes. Two postoperative patients with impairment of voice were observed. One had laryngeal edema following a transfusion reaction; the other, following thyroidectomy with temporary paralysis of the recurrent laryngeal nerve. There were 8 whose dysphonia was due to benign tumors, including 7 with papilloma and 1 with leukoplakia. There were 7 patients with cancers, 2 with carcinoma of the larynx and 2 with secondary invasion of this structure from the esophagus and the thyroid gland. The three remaining patients had lymphosarcoma, leukemia and Hodgkin's disease with associated laryngeal lesions.

TREATMENT OF MISCELLANEOUS GROUP

Medical Treatment.—This consisted in:

1. Treatment of the underlying pathologic process present.
2. Endocrine therapy in cases with congenital malformations and sexual arrest.
3. Physical therapy in cases of thyroidectomy with paralysis of the recurrent laryngeal nerve. It included heat, gentle massage and electrical stimulation.

Surgical Treatment.—This consisted in:

1. Excision of tumors—benign or malignant, with roentgen irradiation of primary growths if these were radiosensitive.
2. Laryngectomy, tracheotomy, etc., if the cause of dysphonia was a cancer.
3. Dilation of scar excision for cicatricial contracture.
4. King, Kelly, Hoover, and other surgical procedures for relief of bilateral abductor paralyses.

Vocal Training.—This is subject to variation depending on the primary problem present:

1. Vocal rest.
2. Exercises to obtain the optimum pitch.

3. Breathing exercises.
4. Mechanical larynx (after laryngectomy).
5. Esophageal speech (after laryngectomy).

Other procedures already recorded are to be carried out as indicated in this group, such as psychotherapy, recordings, etc.

SUMMARY

The various types of dysphonia that were observed in the speech clinics at three Army general hospitals (Brooke, McGuire and Valley Forge) over a period of more than three years are presented. Seventy-eight cases in all were studied, 54 per cent of which resulted from injuries or exposure to circumstances of combat. Owing to the adjacency of many vital structures, almost any combination of traumas of blood vessel, cervical cord and cranial or sympathetic nerves may occur, as illustrated in this series. The methods of examining and treating patients for impairment of voice under different circumstances are described in detail. Actual therapy is best carried out by a speech pathologist well grounded in psychologic technics, under the supervision of the responsible physician. Amelioration of the symptoms commonly followed corrective therapy, often whether or not there was continued evidence of unilateral abductor paralysis or intrinsic scar contracture.

Medical Arts Building.

ROLE OF PENICILLIN IN THE MANAGEMENT OF ACUTE AND CHRONIC FRONTAL SINUSITIS

KENNETH M. KAHN, M.D.*
STATEN ISLAND, N. Y.

AS EARLY as July 1943, at Halloran General Hospital, Champ Lyons and his co-workers had definitely shown the marked bacteriostatic effect of penicillin in suppurating wounds infected with streptococci and staphylococci. Because of the good results being obtained by them, it was decided to use this antibiotic substance in the treatment of suppurative frontal sinusitis.

The cases to be presented were selected because they represent the ones that were the more serious from a prognostic point of view. The degree of pathologic involvement presented varied from acute, nonsuppurative, uncomplicated to chronic suppurative sinusitis with intracranial complications.

Because certain strains of gram-positive micro-organisms were known to be resistant to penicillin, great effort was made to select those cases in which bacterial studies of penicillin sensitivity were possible or in which clinical experience, from a bacteriologic standpoint, gave reasonable assurance of excellent results. Tests of sensitivity were done in all cases in which positive cultures could be obtained.

My co-workers and I are fortunate in that to this date we have not seen any case of suppurative disease of the frontal sinus in which the causative micro-organism was penicillin resistant. This has not been our experience with other types of suppurative diseases in our field.

It is my opinion that, although the results obtained in the cases presented are uniformly excellent, penicillin alone is not the answer to the problem of the management of suppurative diseases of the frontal sinuses. On the contrary, the data gathered here would indicate that our results are similar to those of other investigators. The use of penicillin in the management of chronic suppurative sinus disease leads us, more and more, to feel that it is but an excellent adjunct to the adequate and well planned surgical procedure.

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*Formerly Colonel, Medical Corps, Army of the United States.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc.

REPORT OF CASES

CASE 1 (diagnosis: frontal sinusitis of the right side, acute, nonsuppurative, severe).—K. M. K. was admitted to Halloran General Hospital, April 5, 1945, with a chief complaint of severe pain over the right frontal area and behind the right eyeball, with severe lacrimation of the right eye. The nasal chambers were clear; there was no pus or polyp; the right conjunctiva was congested. There was slight puffiness of the supraorbital area and of the right upper eyelid. Gentle tapping of the frontal area caused severe pain. The upper inner angle on the right side was exquisitely tender to light palpation. The temperature was normal. The patient stated that he experienced a sudden onset of pain twelve hours previously. The blood count and the urinalysis gave normal values.

Administration of penicillin was started immediately, 25,000 units of penicillin being given every three hours; a 1 per cent solution of "neo-synephrine hydrochloride" was used as a nasal spray every three hours. The following day the patient showed considerable improvement. There was still some pain, with tenderness to palpation. The improvement continued until the fourth day, at which time there was no longer any swelling of the eyelid or of the prefrontal area, nor was there any tenderness to palpation. At no time had there been any purulent nasal discharge; therefore, bacterial culture was not possible. Penicillin was discontinued on the fifth day, and the patient was allowed to be out of bed.

Forty-eight hours after the discontinuance of the administration of penicillin, the patient had recurrent pain in the right frontal region, of lesser severity than that on admission. Tenderness to palpation and pain on gentle tapping of the frontal area had returned. For the second time, the patient was given penicillin, 25,000 units every three hours. Twenty-four hours later the recurrent pain and tenderness had disappeared. The use of penicillin was continued over a period of six days. The patient made an uneventful recovery and was returned to full duty.

Comment.—This case illustrates acute nonsuppurative frontal sinusitis responding to penicillin therapy without surgical intervention. It appears that the dosage was adequate but not sustained over a sufficient period to prevent recurrence. Subsequent therapy was necessary to produce the desired result.

It is reasonable to assume, from this case, that adequate and sustained penicillin therapy can be effective in acute frontal sinusitis without surgical intervention if instituted before suppuration occurs.

CASE 2 (diagnosis: frontal sinusitis of the right side, chronic, suppurative, uncomplicated).—A. F. H. was admitted to Halloran General Hospital on May 17, 1944. On March 15, while on duty in England, she contracted a severe infection of the upper respiratory tract. There was complete nasal obstruction, accompanied by profuse purulent nasal discharge, which was still present on her admission to Halloran General Hospital. Severe right frontal headache and tenderness to palpation became evident during the latter part of April. Chills and fever were absent, but she complained of a constant nonproductive cough.

Clinical examination revealed a thick white purulent nasal discharge. In the region of the nasofrontal duct there was a polyp of moderate size which was obstructing drainage from the right frontal sinus. Bacterial culture was positive for hemolytic streptococci of the beta type. The organisms were penicillin sensitive. Roentgen examination of the sinuses showed marked clouding of the right maxillary antrum and the right frontal sinus. The left frontal sinus and the left antrum were

clear. The leukocyte count was 10,450, of which 87 per cent were polymorphonuclears. Urinalysis gave results within normal limits.

The patient was given 25,000 units of penicillin every three hours, beginning May 22. Three days later the purulent discharge had become mucoid. Radical frontoethmoidectomy and intranasal antrotomy were performed on May 26. The right frontal sinus was found to be filled with pus and polyps. The anterior ethmoid cells were filled with pus. They were exenterated, exposing the face of the sphenoid bone. The condition of the mucous membrane of the sphenoid sinus appeared to be within normal limits and was not removed. The anterior tip of the inferior turbinate was removed and the antrum entered. The lateral nasal wall was removed with a biting forceps and the mucous membrane removed so far as possible with Coakley curets. The antrum and the frontoethmoid cavities were packed with one-half inch plain gauze saturated with regular strength penicillin. The wound was closed with interrupted fine silk sutures. Ten thousand units of penicillin was instilled into the packed sinus cavity each day.

The sutures and the packing were removed on the fourth postoperative day. The maxillary antrum and the frontoethmoid cavity were irrigated every second day. The intramuscular injection of penicillin was discontinued on May 30. On

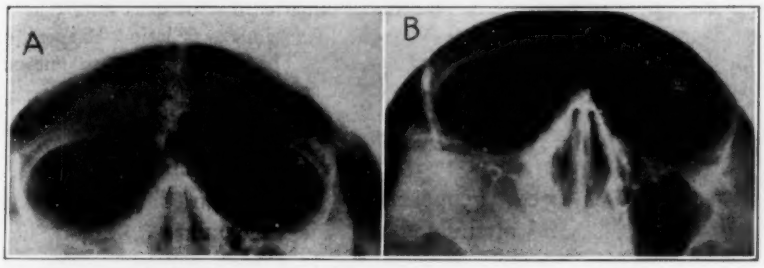

Fig. 1 (case 2).—*A*, preoperative roentgenogram of the right frontal sinus showing complete occlusion and an area of possible osteomyelitis.
B, postoperative roentgenogram showing removal of the disease process.

June 8 roentgen examination of the sinuses revealed a bone defect overlying the right anterior ethmoid cells. The right antrum was less cloudy than on previous examination.

Comment.—This case illustrates a frank purulent disease of the frontoethmoid sinuses with inadequate drainage. The removal of the intranasal polyp could not reasonably assure complete drainage of the frontal sinus. A similar case in which the patient was treated by this method in 1941 resulted in death from abscess of the brain and infection of the blood stream. Surgical intervention alone may have resulted in uneventful recovery, but we feel that the administration of penicillin resulted in a period of protection during which a radical operation was performed without development of postoperative complications.

CASE 3 (diagnosis: frontal sinusitis of the left side, chronic, suppurative, uncomplicated).—R. E. M., was admitted to Halloran General Hospital on Aug. 30, 1943. He had had a trephine opening of the left frontal sinus for acute suppurative disease on June 10 at a station hospital in Africa. He had experienced the usual

train of symptoms associated with acute suppurative frontal sinusitis during the latter part of May. He was evacuated to the United States for definitive care because purulent drainage of the external wound was still present six weeks after operation.

Frontal tenderness and external drainage were present on admission to the hospital. Bacterial culture revealed Staphylococcus aureus hemolyticus, which was shown to be penicillin sensitive. Penicillin therapy, 25,000 units every three hours, was instituted. The purulent discharge became mucoid, and the frontal tenderness disappeared in four days.

Radical frontal sinusotomy was performed on September 3. The previous opening, 1 cm. in diameter, was noted. This opening was enlarged with rongeur and the inferior third of the anterior plate of the frontal sinus removed. The entire sinus was filled with infected granulations. These were removed with curets. Anterior ethmoidectomy was performed for adequate intranasal drainage. There was no definite clinical evidence of osteomyelitis. The frontal sinus was loosely packed with selvage edge sulfathiazole gauze and the wound closed with interrupted silk sutures. A small rubber drain was placed in the sinus at the inner angle. The packing was removed through the nose on the fourth postoperative day and the sinus irrigated with isotonic solution of sodium chloride. The patient was given intramuscular injections of penicillin for a period of four days after operation. Local application of penicillin was not used. The sinus was irrigated every third day with isotonic solution of sodium chloride. The patient made an uneventful recovery and was returned to full duty on September 21.

Comment.—This case illustrates chronic purulent infection of the frontal sinus without adequate drainage. A penicillin-sensitive micro-organism was cultured preoperatively. Penicillin therapy alleviated frontal tenderness and attenuated the causative micro-organism within four days. Complete recovery followed radical surgical intervention more promptly than had been our experience with patients observed and treated prior to the advent of penicillin.

CASE 4 (diagnosis: frontal sinusitis of the right side, chronic, suppurative; acute exacerbation with acute osteomyelitis of the frontal bone).—G. R. R. was admitted to the Regional Station Hospital at Fort Jay, N. Y., on Sept. 25, 1943. He had a profuse purulent nasal discharge and severe headache in the right frontal area. Exquisite frontal tenderness to light palpation and retro-ocular pain were present. His temperature was not elevated. There was increased tenderness over the right frontal area, with localized edema, on September 29. There was an increase of intranasal discharge. A trephine opening was made in the floor of the right frontal sinus. Incision of the mucous membrane protruding through the trephine opening resulted in the recovery of frank pus. Bacterial culture of the pus was positive for nonhemolytic streptococci.

The patient was transferred to Halloran General Hospital on November 3. Examination of the wound revealed a copious amount of pus exuding from the right frontal sinus, with marked edema of the supraorbital soft tissues. There was exquisite tenderness to palpation. The temperature was normal and the pulse rate 80. The blood pressure was 115 systolic and 60 diastolic.

There was increased edema of the supraorbital area two days later. Bacterial culture revealed penicillin-sensitive nonhemolytic streptococci. Penicillin therapy, 25,000 units every three hours, was begun. The patient felt better within forty-

eight hours. The edema over the right frontal area had definitely subsided, and the frontal tenderness had disappeared. The profuse purulent drainage was decreased and beginning to change to a mucoid character.

Roentgen examination showed homogeneous occlusion of the right antrum and of the frontal and ethmoid sinuses. The border of the right frontal sinus was only faintly visible. There was no evidence that bone had been destroyed. The blood count showed 9,000 white cells, with 53 per cent polymorphonuclear leukocytes and 44 per cent lymphocytes. Urinalysis gave normal results.

The patient was seen in consultation with Major Lyons on November 11, who felt that the time was propitious for surgical intervention. Radical frontal sinusotomy was performed on November 12. Large amounts of mucoid material and polypoid tissue were observed within the frontal sinus. A sequestrum was found involving the inner surface of the outer table toward the midline. The incision was elongated by extension across the root of the nose. The outer table was explored from the under surface and the devitalized bone removed by curet. The outer surface of the outer table was not affected. Anterior ethmoidectomy was

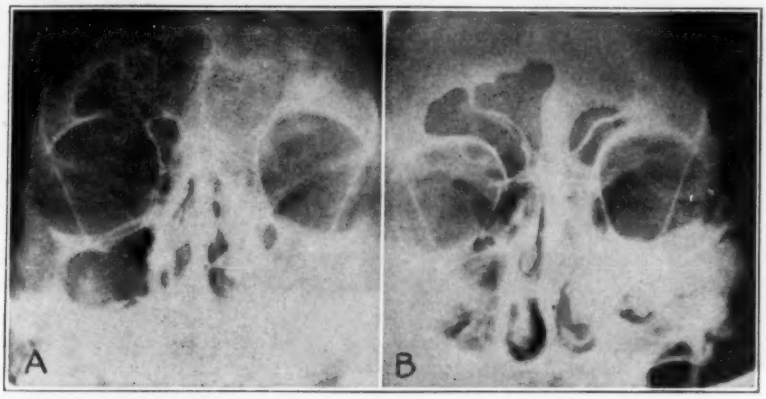

Fig. 2 (case 4).—*A*, preoperative roentgenogram showing complete occlusion of the right frontal sinus.

B, postoperative roentgenogram showing operative defect in frontal bone.

performed for adequate intranasal drainage. The sinus cavity was packed with selvage edge gauze saturated with regular strength penicillin. The wound was closed with interrupted fine silk sutures and a small rubber drain placed in the inner angle of the incision.

The sinus packing and the sutures were removed on the fifth postoperative day and the frontal cavity irrigated every three days postoperatively until December 4. The intramuscular injection of penicillin was continued until November 23.

Roentgen examination of the sinuses, December 13, showed postoperative changes over the right frontal sinus. The granular-appearing bone had been removed, and no sequestrums were seen.

Comment.—This case presents a suppurative process of the frontal sinus complicated by acute osteomyelitis of the frontal bone. The offending micro-organism proved to be penicillin sensitive. Adequate penicillin therapy controlled the pathologic spread of the osteomyelitis

and produced attenuation of the causative micro-organism. Optimum conditions under which a radical operation could be performed were afforded, enabling one to arrive at the desired end result.

CASE 5 (diagnosis: frontoethmoid sinusitis, acute, suppurative, complicated by meningitis).—J. L. B. was admitted to Halloran General Hospital on April 4. He was in coma, with a rigid neck and a temperature of 106.4 F. There were intense left orbital cellulitis and exquisite tenderness over the left frontal sinus. Kernig and Brudzinski signs were present. The left nasal chamber contained a small amount of pus from which on culture Staph. aureus hemolyticus grew, which was penicillin-sensitive. The blood showed leukocytosis, the count being 14,500, 81 per cent of which were neutrophils. The urine contained an occasional granular cast and 3 to 5 red blood cells per high power field. Spinal tap was unsuccessful because of the irrational condition of the patient.

Penicillin therapy, 50,000 units every three hours, was immediately instituted. Twenty-four hours later the patient appeared slightly improved. He was more rational, the orbital cellulitis was slightly improved and his temperature had dropped to 105 F. The temperature continued to decrease, reaching 104.2 F. on the second day, but the meningitic signs did not improve. Roentgen examination showed disease of the left frontal and ethmoid sinuses.

Spinal puncture was first successful five days after admission. The cerebrospinal fluid was under a pressure of 310 mm. of mercury and contained 1,350 leukocytes per cubic millimeter. The spinal fluid sugar was 37 mg. per hundred cubic centimeters. Fifteen thousand units of penicillin was injected intrathecally. Roentgen examination revealed an increase of pathologic change over that observed in the previous examination.

Radical frontoethmoidectomy was performed on April 10. A spinal puncture was made immediately before the operation. The cerebrospinal fluid revealed an increase in leukocytes to 2,300 per cubic millimeter and a decrease in sugar to 17 mg. per hundred cubic centimeters. The frontal sinus was filled with creamy pus and polyps; the ethmoid cells, with a reddish gelatinous material, under tension. The sphenoid sinus was normal. Two no. 5 ureteral catheters were placed into the common cavity for local instillation of penicillin—one into the frontal sinus and the other into the ethmoid labyrinth. They were anchored to the skin in the angles of the external wound.

Twenty-four hours after operation the patient's general condition was improved, and within four days his temperature had declined to 100 F. Packing, sutures and catheters were removed on the fourth postoperative day and the local application of penicillin discontinued. Spinal puncture, with intrathecal administration of penicillin, was continued daily until the ninth postoperative day. The spinal fluid then showed 52 lymphocytes per cubic millimeter, a pressure of 180 mm. and a sugar content of 52 mg. per hundred cubic centimeters.

Intramuscular injection of penicillin was discontinued April 30. The patient was up and about the ward. Final spinal tap, urinalysis and blood count gave results within normal limits. The patient was discharged from the hospital after he had been given a ninety day convalescence furlough.

Comment.—Immediate institution of adequate penicillin therapy produced clinical improvement in this case. The pathologic process advanced, however, which was substantiated by laboratory and roentgen findings.

We are of the opinion that this case would have terminated fatally if radical surgical intervention had not been accomplished.

CASE 6 (diagnosis: frontal sinusitis of the left side, chronic, suppurative).—M. M. B. was admitted to Halloran General Hospital, Feb. 23, 1945, for phthisis bulbi of the left side, due to injury incurred in enemy action. He was seen in consultation because of a discharging sinus just above the inner canthus of the left eye. He stated that this draining sinus had been present since he was injured in enemy action on January 3, but there had been no surgical intervention.

On March 1 he was seen by the internists because of acute hepatitis. It was advised that any operative procedure be postponed. The patient remained on the

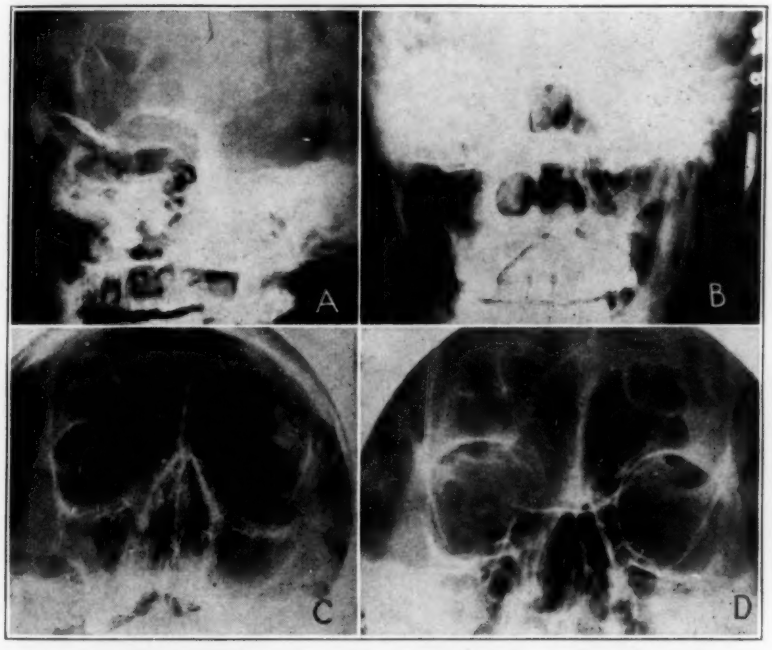

Fig. 3 (case 5).—*A*, oblique view showing disease of the ethmoid cells.

B, posterior view showing disease of the left posterior ethmoid and frontal sinuses. The left sphenoid sinus appears occluded.

C, postoperative view of the left frontal sinus showing removal of the disease process.

D, postoperative posterior view showing clearing of the frontal and ethmoid sinuses.

Medical Service until April 17, at which time he was transferred to the Eye Service. The patient was seen again, and it was felt that an exploratory sinusotomy should be done. The bacterial culture was negative, but roentgen examination confirmed the diagnosis of chronic suppurative left frontal sinusitis.

External frontoethmoidectomy was performed on April 18. The patient was given 500 cc. of whole blood during the operation. Advanced pathologic change was found in both the frontal sinuses and in the left ethmoid sinus. The floor and

the inferior third of the external frontal lamina were osteomyelitic. The left internal lamina was completely necrotic, with the left orbital fascia fused to the dura over the frontal lobe.

The supraorbital ridge and all infected polypoid tissue of the frontal sinuses were removed. The interfrontal septum was not present. We were therefore able to exenterate the contents of the right frontal sinus through the left frontal wound. The lacrimal bone and the lamina papyracea of the ethmoid bone on the left side were completely gone. The orbital fascia was carefully incised and separated from the dura. Anterior ethmoidectomy was performed through the nose and the external wound for adequate intranasal drainage. The frontal and ethmoid sinuses were packed with one-half inch gauze saturated with penicillin, and two no. 5

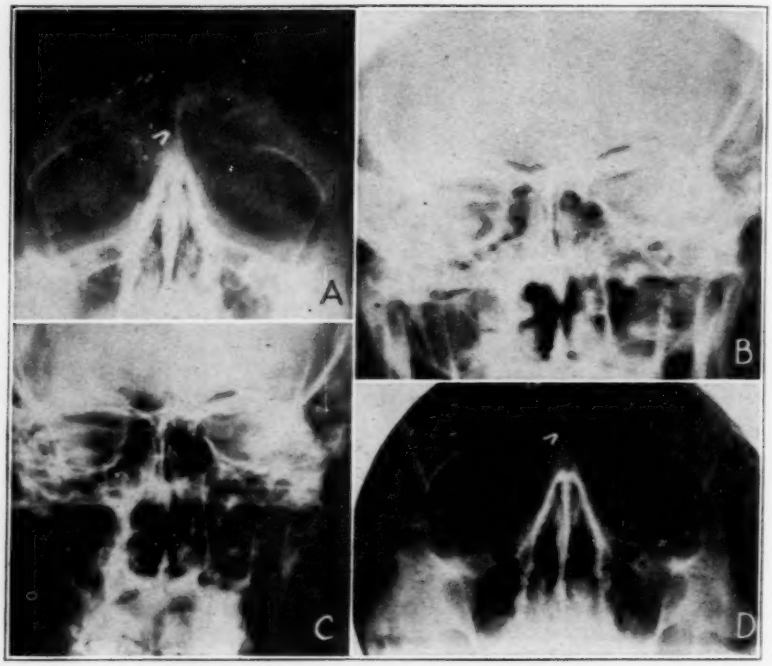

Fig. 4 (case 6).—*A*, preoperative Waters view showing disease of the frontal sinuses with destruction of the left supraorbital ridge and of the anterior ethmoid cells.

B, preoperative posterior view of sinuses showing disease of the frontal sinuses and of the left ethmoid sinus.

C, postoperative posterior view showing operative defect in the left frontal bone with clearing of the clouded left posterior ethmoid cells.

D, postoperative view of the left frontal sinus.

urethral catheters were anchored in the upper and lower poles of the wound. One was directed into the right frontal sinus and the other into the ethmoid cavity. The wound was then closed with interrupted fine silk sutures.

On the following day the patient was sitting up in bed, eating a soft diet. The wound was dressed on the fourth postoperative day. The packing and the urethral catheters were removed, and the local use of penicillin was discontinued.

The sutures were removed on the fifth day and the sinus cavity irrigated with isotonic solution of sodium chloride. Irrigation was repeated every third day for a period of twelve days.

No further treatment has been necessary. At the present time the patient is still receiving ophthalmic plastic reconstruction, but he is completely free of otolaryngologic symptoms.

Comment.—This patient did not require penicillin before operation because the discharge from the frontal sinus was minimal and the culture negative for micro-organisms. A radical surgical procedure and local

Fig. 5 (case 6).—Postoperative photograph of the patient showing the sinus and postoperative incision healed. The left bulbus oculi is an acrylic prosthesis.

instillation of penicillin were used with primary closure of the operative wound. Healing occurred within four days. This case illustrates the excellent local efficiency of penicillin in promoting rapid healing of wounds.

CASE 7 (diagnosis: osteoma of the frontal sinuses on which is superimposed acute suppurative frontal sinusitis).—R. L. C. was admitted to the Regional Station Hospital at Fort Jay, N. Y., Feb. 1, 1945, with a history of headaches since 1941. Roentgenograms of the sinuses taken in 1942 revealed a large osteoma involving the frontal sinuses. Four days previous to admission to the Station Hospital he complained of a severe frontal headache. It was constant and unrelieved by

medication. There was a profuse nasal discharge with a postnasal drip. A blood count showed 18,050 leukocytes, 82 per cent of which were neutrophils. The urine was normal. Penicillin therapy, 25,000 units every four hours, was instituted.

The next day considerable edema and tenderness were present over the frontal sinuses. His temperature had risen to 102 F. The diagnosis of acute osteomyelitis

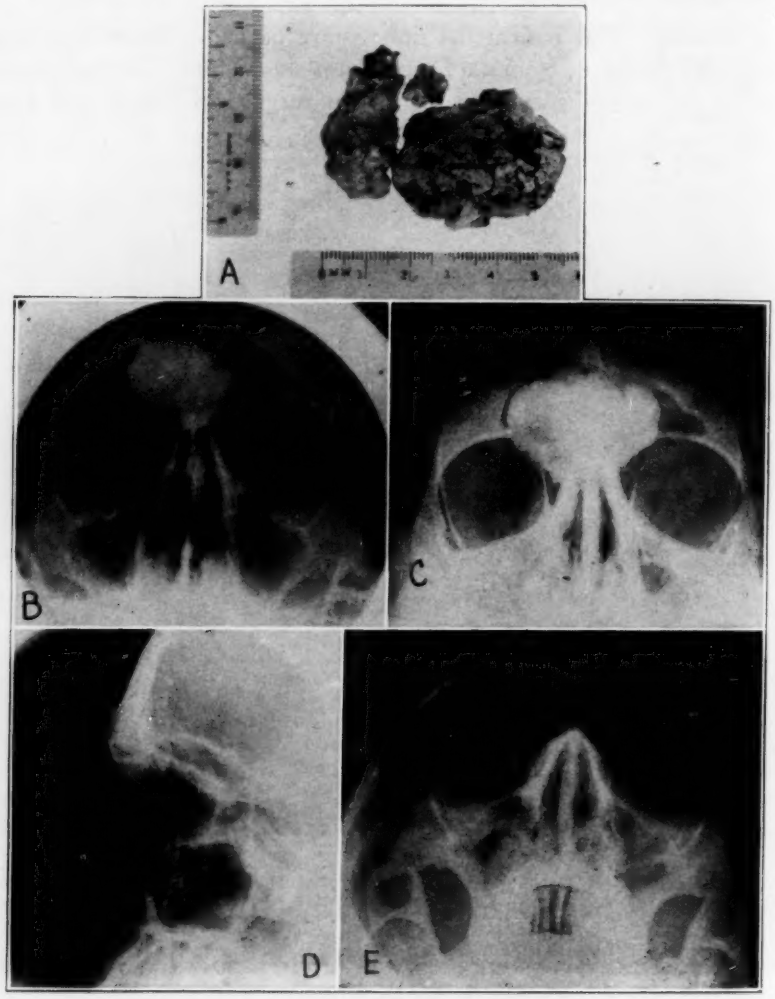

Fig. 6 (case 7).—*A*, osteoma after surgical removal.

B, osteoma of the frontal sinus as observed in 1942, without superimposed acute frontal sinusitis.

C, progressive enlargement of the osteoma with suppuration as noted in 1945.

D, lateral view showing almost complete filling of the sinuses.

E, postoperative appearance of the frontal sinuses ten days after removal of the osteoma.

of the frontal bone was made. Immediate drainage of both frontal sinuses was accomplished by trephination. The osteoma was found to be in direct contact with

the anterior walls and floors of the frontal sinuses. Pus was encountered on both sides but was more profuse on the left. Culture of the recovered pus revealed no bacteria.

On February 4 the patient's temperature was normal. The general condition was much improved, and the external drainage had lessened considerably. By February 19 the patient's condition had improved to such a degree that penicillin therapy was discontinued. By telephone Major Doust was consulted as to the future disposition of the patient. He was transferred to Halloran General Hospital for definitive care on March 3.

On admission the patient was not acutely ill. There was an external draining sinus in the region of the inner frontal angles. Review of roentgenograms showed that the osteoma had increased greatly in size from 1942 to 1945; it almost com-

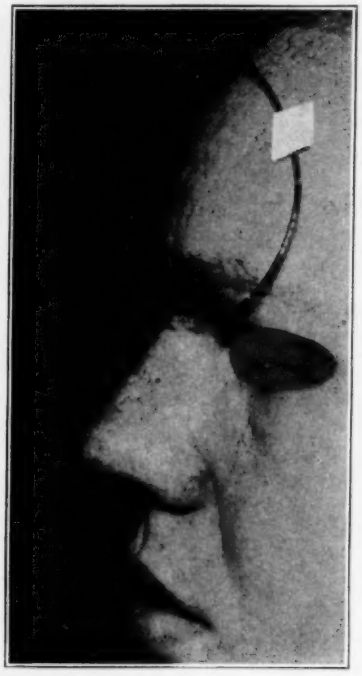

Fig. 7.—Ureteral catheter shown in situ on the first postoperative day, for the local instillation of penicillin.

pletely filled the frontal sinuses. In addition, there was roentgenologic evidence of advanced disease of the sinuses lateral to the osteoma.

Culture of the frontal sinus drainage revealed hemolytic streptococci of beta type, which were penicillin sensitive. Penicillin therapy, 25,000 units every three hours, was started on March 12. Forty-eight hours later the discharge had diminished almost completely and was of a mucoid character.

Bilateral radical frontal sinusotomy, with removal of osteoma, was performed on March 14. The operation was accomplished through a bilateral Killian incision connected across the root of the nose. Exposure of the frontal sinus revealed erosion of the anterior lamina. The external plate was removed with mallet and gouge, exposing the osteoma, in mass. The base of the osteoma was tunneled

Summarization of Therapeutic Procedures in Seven Cases of Frontal Sinusitis

Diagnosis	Duration	Organism	Preoperative Penicillin Therapy		Operative Procedure	Postoperative Penicillin Therapy		Length of Observation	Results
			Duration	Dosage		Duration	Dosage		
Case 1. K. M. K. ..... Acute nonsuppurative frontal sinusitis, right	½ day	None obtained	10 days	25,000 units every 3 hr.	None	.....	.....	16 days	Cure
Case 2. A. F. H. ..... Chronic suppurative fronto-ethmoiditis, early, right	2 mo.	Hemolytic streptococci, beta type	4 days	25,000 units every 3 hr.	Radical external frontosphenoeth-moidectomy, right	4 days	25,000 units every 3 hr.	1½ mo.	Cure
Case 3. R. E. M. ..... Chronic suppurative frontal sinusitis, left	3 mo.	Hemolytic Staph. aureus	4 days	25,000 units every 3 hr.	Radical frontal sinusotomy, left	4 days	25,000 units every 3 hr.	2¾ mo.	Cure
Case 4. G. R. R. ..... Chronic suppurative frontal sinusitis; acute exacerbation with osteomyelitis of frontal bone	2 mo.	Nonhemolytic streptococci	7 days	25,000 units every 3 hr.	Radical frontal sinusotomy, right	11 days	25,000 units every 3 hr.	1½ mo.	Cure
Case 5. J. L. B. ..... Acute suppurative frontosphenoethmoiditis with orbital cellulitis and meningitis	1-3 days	Hemolytic Staph. aureus; hemolytic streptococci	4 days	50,000 units every 3 hr.	Radical fronto-sphenoethmoi-dectomy, left	Locally 4 days Intrathecally 8 days Intramuscularly 21 days	10,000 units every 3 hr. 15,000 units every 3 hr. 50,000 units every 3 hr.	5 mo.	Cure
Case 6. M. M. B. ..... Chronic suppurative frontoeth-moiditis with chronic osteo-myelitis of frontal bone	3½ mo	None obtained		None	Radical external frontoethmoi-dectomy	Locally 4 days	20,000 units every 3 hr.	5 mo.	Cure
Case 7. R. L. C. ..... Acute suppurative frontal sinusitis with large osteoma of frontal sinuses	14 days	Hemolytic streptococci, beta type	2 days	25,000 units every 3 hr.	Bilateral radical frontal sinusot-omy, removal of osteoma	Locally 4 days Intramuscularly 9 days	10,000 units every 3 hr. 25,000 units every 3 hr.	2 mo.	Cure

through in the region of the root of the nose. The upper portion was removed by rocking it with grasping forceps. The base was then removed with biting forceps. It measured 5 by 3 by 2 cm. All polyps and infected mucous membrane were removed with Coakley curets. The medial halves of the supraorbital ridges were necrotic. They were removed with biting forceps. Bilateral anterior ethmoidectomy was performed for adequate intranasal drainage. The margins of the external frontal lamina were smoothed down in order that a tantalum plate might be received more easily at a later date. The frontal cavity was packed loosely with 1 inch (2.5 cm.) plain gauze saturated with regular strength penicillin. Two no. 5 ureteral catheters were placed within the frontal sinuses, for the local instillation of penicillin, and anchored by suture in the outer angles of the wound. The external wound was approximated with multiple interrupted mattress silk sutures.

Four days later the sutures and the ureteral catheters were removed and the local instillation of penicillin was discontinued. The intramuscular injection of penicillin was continued until March 23. On March 20 the patient's condition was excellent. There was almost no intranasal discharge. The frontal cavity was irrigated with isotonic solution of sodium chloride. On March 25 and 30 the irrigation return was clear. The patient was given a convalescence leave on April 3. He returned to limited duty May 2.

Comment.—This case presents an osteoma of the frontal sinuses obstructing normal drainage. An acute suppurative infection complicated this condition, resulting in acute fulminating osteomyelitis of the frontal bone. The bacteriostatic action of adequate and sustained penicillin therapy controlled the acute phase of the disease, but in our opinion radical surgical intervention was absolutely necessary to obtain complete recovery.

SUMMARY

Seven cases of frontal sinus disease have been presented in which penicillin was used in an effort to determine the value of its use in comparison with those chemotherapeutic measures available to us before its advent. Its use has been by local instillation, intrathecal or intramuscular injection or a combination of these methods. The causative micro-organisms were tested for penicillin sensitivity when this was possible. Penicillin-resistant organisms were not encountered. The degree of pathologic involvement varied from acute nonsuppurative to chronic suppurative sinusitis with intracranial complications. The desired end result was obtained in each case which came under observation.

In a case of acute nonsuppurative frontal sinusitis complete recovery was obtained without surgical intervention (case 1). In all cases of suppurative sinusitis, whether acute or chronic, adequate surgical intervention was necessary in order to produce complete recovery. Bacteriostasis was secured in acute fulminating disease. This permitted adequate surgical intervention without further dissemination of the pathologic process, with the desired end results.

CONCLUSIONS

1. Penicillin is a powerful antibiotic substance which, by its bacteriostatic properties, attenuates certain gram-positive cocci and thus affords a period of protection during which radical surgical intervention may be accomplished without the development of the postoperative complications encountered before its introduction.

2. Once suppuration has occurred, penicillin therapy must be supplemented with adequate surgical intervention in order to obtain complete eradication of the pathologic process.

3. We feel justified in believing that instilling this antibiotic drug in postoperative wounds affords primary closure and thereby decreases to a marked degree the usual period required for complete healing.

15 Beach Street.

INHALATION OF PENICILLIN AEROSOL AND PENICILLIN-STREPTOMYCIN AEROSOL IN OFFICE PRACTICE

DAVID DAVIS, M.D.
WASHINGTON, D. C.

PENICILLIN aerosol was given a fairly extensive trial in the treatment of patients with infections of the respiratory tract in my office during the past two years. There were two purposes in mind: (1) to relieve the patient of his illness and (2) to do it through office treatments rather than through hospitalization. At the beginning of my studies on penicillin aerosol the only other available methods of administering penicillin were those of intramuscular and intravenous injection, which required hospitalization. Penicillin in oil and wax was not as easily obtained as it is today and therefore could not be used with any regularity.

I attempted to find out the effect of penicillin aerosol in the usual run of patients that were seen in the average otolaryngologist's office, and I tried it on any patient in whose case it seemed logical that penicillin applied locally would be of value. The patients included those with acute and also some with chronic infections of the upper and lower respiratory tract. In fact, when I first began to use the penicillin aerosol, all of the patients selected had chronic conditions—bronchiectasis, chronic bronchitis and bronchial asthma. Then I used it on those with acute bronchitis, sinusitis, nasopharyngitis, laryngitis, tracheitis and coryza. The technic outlined by Barach and associates [1] and Vermilye [2] was followed, with modification as the occasion required.

TECHNIC

At the outset some patients were given an inhalation every hour for four to six treatments a day, 5,000 to 50,000 Oxford units being used each time; as time progressed the number of treatments was decreased and the number of units increased until now one treatment is given daily, with 100,000 units in 1 cc. of

From the Department of Otolaryngology, George Washington Medical School.

1. Barach, A. L.; Silberstein, F. H.; Oppenheimer, E. T.; Hunter, T., and Sorokee, M.: Inhalation of Penicillin Aerosol in Patients with Bronchial Asthma, Chronic Bronchitis, Bronchiectasis and Lung Abscess: Preliminary Report, Ann. Int. Med. **22**:485 (April) 1945.

2. Vermilye, H. N.: Aerosol Penicillin in General Practice, J. A. M. A. **129**:250 (Sept. 22) 1945.

salt solution.[2a] Case histories will be given later. In general it can be stated that the results were gratifying and worth while. Another factor which may be of some value to the physician is that the treatment can be given by the office nurse, once the examination and the diagnosis are made. The daily check-up takes only a minute of the physician's time, the nurse carrying on from there. The patients will tell the nurse more frankly whether the treatment is making them feel better or not, and a record is kept of their statements and also of the physical findings.

The apparatus used at first was a DeVilbiss nebulizer, no. 40, connected by a rubber tube with a large oxygen tank fitted with a reducing valve. The near end of the nebulizer was held in the mouth or was fitted with two tips which were placed in the nasal vestibules. From 5 to 6 liters of oxygen per minute

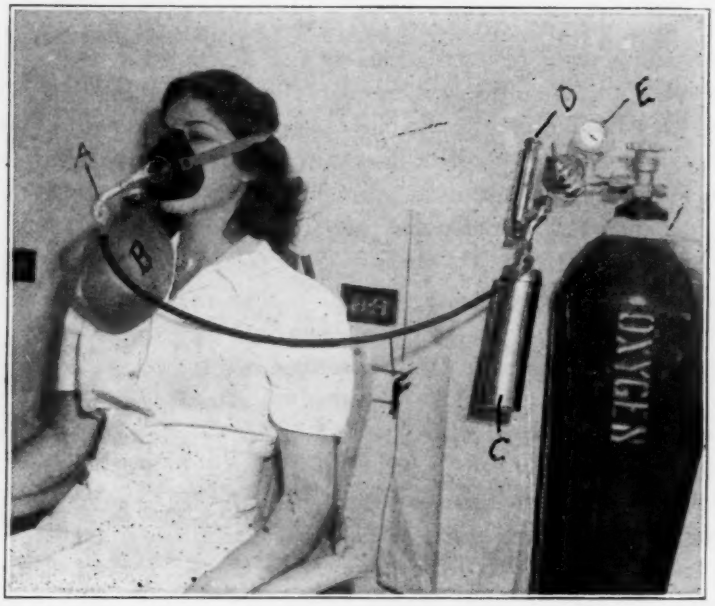

Fig. 1.—Patient using the mask with rebreathing bag: *A*, nebulizer; *B*, rebreathing bag; *C*, humidifier; *D*, gage to measure liters of oxygen per minute; *E*, gage to show amount of oxygen in tank. A Y tube can be inserted in the rubber tube, *F*, and the patient instructed to close the open end of the Y tube with his finger on inhalation and to open it on exhalation.

2a. I now use a combination of penicillin and streptomycin 100,000 units of each, instead of penicillin alone; the antibiotics are dissolved in propylene glycol. This combination is much more effective than penicillin alone in salt solution. However I have been gratified by the good response to penicillin alone, and at times I use it in propylene glycol without the streptomycin. The combination of propylene glycol and either penicillin or streptomycin or both is more effective than that of salt solution and the antibiotics. "Propylene glycol . . . not only is a good vehicle for penicillin aerosol but serves as an adjunct in increasing the antibiotic properties of the blood" (Prigal, S. J.; McGavack, T. H.; Speer, F. D., and Harris, R.: J. A. M. A. **134**:932 [July 12] 1947).

flowed through the nebulizer into which the penicillin[3] in salt solution was placed. Now a face mask is used instead of a mouth piece, and a nebulizer with a rebreathing bag is attached (fig. 1). For sinus treatments a recently acquired nasal tip apparatus is being tried out (fig. 2), which allows suction and pressure so that the penicillin may enter into the sinuses. The patient sits in an ordinary treatment or office chair while taking the treatment, which lasts from ten to fifteen minutes, and is comfortable. Most children are cooperative once they find that the mask does not bother them. The number of treatments depends on the chronicity of the condition. Patients with acute coryza or acute sinusitis usually respond after two

Fig. 2.—Patient using another type of apparatus. This apparatus creates a vacuum when the patient holds her breath and pulls down the handle, *D*. The technic is quite simple: 1. Inhale through the nose and exhale through the mouth for three or four breaths. 2. At the peak of inspiration of the third or fourth breath, close the mouth and exhale through the nose. This raises the pressure in the nose to 20 mm. of mercury and carries the air and penicillin aerosol into the sinuses. 3. Inhale and exhale as in direction 1 for three or four more breaths and at the peak of inspiration of the fourth breath swallow or otherwise raise the soft palate while shifting the handle, *D*, to the horizontal position for negative pressure. This will draw air from the sinuses. After four or five seconds raise the handle, *D*, to the vertical position. This relieves the negative pressure, and air reentering the sinuses will carry penicillin aerosol with it. The degree of negative pressure is regulated by the rate of oxygen flow. The optimum pressure range will be found with flows of from 7 to 10 liters per minute. 4. Repeat from direction 1.

In my office use is made also of the mask with rebreathing bag (fig. 1) in the treatment of the sinuses, and this is found satisfactory. I am unable to compare the results obtained with the two types of apparatus, as both are satisfactory.

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3. Penicillin preparations supplied by Schenley Laboratories, Inc., were used in this work. At first the sodium salt was used, but now the calcium salt is used, as it is more pleasant to the patient.

treatments, while those with chronic bronchitis or bronchiectasis may require many daily treatments before showing any appreciable effect. However, those with chronic conditions usually state that they feel better, that the secretion is easier to cough up and that they sleep better even after the first few treatments.

SURVEY OF THE RESULTS

A number of the patients have not been helped. Whether because of an insufficient number of treatments or because the organisms were not susceptible to penicillin [3a] or because the penicillin did not reach the infection, some did not respond. In the beginning the doses of penicillin were much smaller and probably the concentration was not great enough. I did notice, however, that even as little as 10,000 units of nebulized penicillin produced a definite response; the same amount injected would have had no appreciable effect. Many patients with acute and chronic bronchitis, pharyngitis and sinusitis reported good

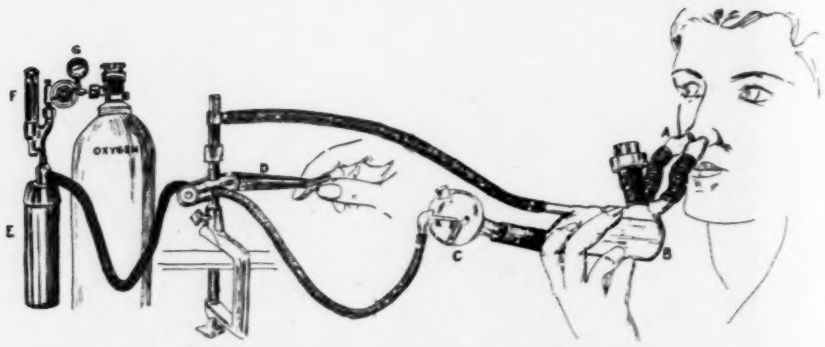

Fig. 3.—Diagram of the apparatus used by patients taking penicillin aerosol for sinusitis (see legend under figure 2): *A*, nasal tips; *B*, nasal trap with pressure control valve; *C*, nebulizer; *D*, handle of valve to create vacuum or pressure; *E*, metal humidifier; *F*, gage to measure number of liters of oxygen per minutes; *G*, gage for oxygen tank.

results from inhalations of penicillin after they had failed to get any relief with intramuscular injections of apparently adequate doses of penicillin.

One of the first patients was a woman 48 years of age who had had a chronic cough since babyhood. She stated that she had tried "every treatment known" and had "even gone to Europe for relief." Roentgen examination showed chronic bronchitis, although bronchoscopy in 1942 (three years before) did not reveal any pathologic change. Iodized oil instilled into the tracheobronchial tree gave partial relief for a few days, but there was always the desire to clear phlegm from her bronchial tubes. When she succeeded, she felt better. There was no pathologic involvement of the sinuses. The lingual tonsil was enlarged and infected and was treated by cautery. Over a period of twenty-eight days she was given 700,000

3a. Cultures of sputum or nasal secretions were made only when the conditions were chronic or when relief had not been obtained by the treatments.

units of penicillin by inhalation. Her reports from the first treatment were that she was not doing any "needless coughing," that "mucus was much thinner and easier to expel," that she was "sleeping better and getting more rest" and that her head felt "so clear and clean." The cough persisted to a much less degree, and general relief was obtained.

Patient 23, a girl 20 years old, had bronchiectasis of many years' duration. Once or twice a day she coughed up a large quantity of mucopus and "coughed a lot at night." Blood agar cultures showed many colonies of Bacillus mucosus capsulatus with a few colonies of Staphylococcus aureus. She was given 250,000 units of penicillin by inhalation over a fourteen day period. She received relief in that the secretion was thinner and she could cough it up with more ease. Also her coughing spells at night were not too severe and thus she obtained more rest than she had for years. The dose in this case was much too small, but these treatments were given before it was realized that larger doses were more efficient. Now she would have received 1,400,000 units in fourteen days, instead of only 250,000.

Patients with laryngitis and tracheitis responded readily to inhalations of penicillin. From one to ten treatments were sufficient in most cases to effect complete relief from hoarseness, cough, dryness and raspy sensations of the trachea and inability to raise mucus.

Patient 7, a physician's wife, who had been a singer, had a cough that had lasted a year, dryness of the throat, a raspy voice which prevented her singing and inability to cough up phlegm which she felt was lodged in her trachea. On Nov. 7, 1945, she inhaled 20,000 units of penicillin, and 50,000 units daily for two more days. The cough diminished, there was less clearing of the throat and she felt better in general. Treatments were interrupted until Jan. 4, 1946, when 100,000 units was inhaled daily for five days. There was almost complete relief of all symptoms; she was able to sing again, and the cough had disappeared completely. The sensation of mucus in the trachea had gone, and the patient was apparently well. Objectively, also, her larynx and trachea had lost their inflammation, and the cords approximated normally.

Asthmatic patients were treated at first without regard to etiologic factors. I have concluded, however, that only those with infectious asthma are helped sufficiently by penicillin aerosol and now confine the treatments to that type. I was gratified in the beginning to find that many patients whose asthma was due to specific noninfectious allergens were helped considerably by penicillin aerosol, apparently by removal of the superimposed infectious factor. In patients with the infectious type, apparently sufficient organisms in the nose, throat and lower respiratory tract were killed off to influence the asthma itself.

Similarly, patients with sinobronchitis received a great deal of benefit, and many of them, apparent cures. Those with acute disease were relieved after one to five treatments, each treatment being the inhalation of 100,000 units of penicillin in 1.5 to 2 cc. of salt solution. Those with chronic disease felt relieved also after a few treatments, but the nasal, postnasal and bronchial secretions continued for a longer period. The patients claimed, however, that the secretions were thinner

and more easily expectorated. Almost all of these patients were able to sleep better because the coughing which usually occurred when they lay down was relieved. Their chests felt clearer, and the need of clearing their throats was lessened. Many also stated that their headaches were relieved and that the sense of pressure in the bridge of the nose disappeared. The secretions often changed from purulent or mucopurulent to mucoid or thinner secretions and were more easily blown from the nose or expectorated. There was a feeling of well-being, and the head and the chest felt lighter.

The number of treatments necessary for these chronic conditions depended on the response. After the patient stated that the condition had definitely improved and that the secretions had been reduced so that they were not annoying, the treatments were discontinued. If symptoms returned, the patient was advised to have another course of inhalations, usually about five or six. Often one course of ten treatments, followed a little while later by another course of six treatments, was sufficient to relieve the patient. The patients were advised to return as soon as a new cold developed, and often one or two inhalations were sufficient to prevent further progress of the infection.

Patients with chronic sinusitis were benefited to the extent that their "heads felt more open and less stuffy" and the secretion was easier to blow out or expectorate. There were fewer exacerbations of acute attacks on top of the chronic condition, and the acute attacks were often abated by one or two inhalations. The intranasal appearance did not change sufficiently to be definite, and there was no effect on any polypi present. The main effects in these patients with chronic sinusitis were the sensation of the nose being clearer, the elimination of the stuffy feeling and the thinning or elimination of the secretions.

Penicillin aerosol will occasionally abort an acute cold of the head or the chest in one or two treatments in spite of the fact that most authors state otherwise. Several patients in whom, following a head cold, a chest cold had always developed that lasted several weeks seemed benefited by one or two inhalations. Apparently the secondary invaders were eliminated, and relief was obtained.

In cases of frank sinusitis, with the nares full of purulent or mucopurulent secretion, a preliminary treatment with nasal sprays and suction was given.

Technic.—"Solution of neo-synephrine hydrochloride, 0.25 per cent," or a solution containing ephedrine sulfate, 1 per cent, is sprayed into both nares, and after a few minutes the secretion is removed by suction, a fine metal cannula being used. (If the nose is merely congested, and no secretion is found, the spray alone is used before the inhalation.) A frank infection of a maxillary sinus, with purulent or mucoid secretion, requires that the sinus be irrigated through the middle meatus or a puncture made through the inferior meatus. This, however, is not usually necessary until the condition has been present for a week or more or is causing pain.

If the patient is seen during the first week of acute sinusitis, inhalations of penicillin will often clear up the condition before punctures are necessary. A daily inhalation of 100,000 units in 1 cc. of salt solution[4] is given, and usually only three to five treatments are necessary to effect a cure. The patients are happy to escape the punctures and are very willing to return daily for the inhalations. If the inhalations fail to effect relief, however, puncture and irrigation are done and the inhalations are continued only if bronchitis is present. The combination treatment shortens the duration of the infection and gives the patients a feeling of well-being which they apparently do not have without the penicillin.

A few patients with nasal allergy due to pollens or dust have been treated by inhalation but without effect. No difference could be seen in the nares after the treatments, and the patients did not obtain any relief. Some benefit was obtained in several instances in which the allergy was complicated by an infection with purulent or mucopurulent secretion. The postnasal discharge in these instances was lessened and the cough eliminated. Also, those patients in whom the lymphoid tissue of the posterior pharyngeal wall would become injected and sore when subjected to the postnasal discharge obtained much relief, and the injection disappeared, although the tissue was not otherwise affected. I believe that some of those patients who showed a favorable response to penicillin had bacterial allergy and reacted similarly to the patients with bacterial asthma. This was especially noticed in children whose previous treatments for these conditions consisted almost wholly in the use of nose drops, with poor results. The incidence of colds, sore throats and cough was materially reduced. The dose for children was 50,000 units of penicillin in 1 cc. of isotonic solution of sodium chloride and there was rarely any difficulty in getting the children to take the treatments. They were happy that their noses did not have to be treated by applications or drops, and their parents were pleased that the treatment could be given almost as soon as they entered the office, since the nurse gave the treatment, thus eliminating the period of waiting to see the doctor. I believe failure to help young patients with nasal disease is often due to inability to do what is necessary for them because of their lack of cooperation. This has been eliminated to a large extent by the inhalations of penicillin.

The ordinary sore throat or acute pharyngitis showed a definite good response to the inhalations, especially when the infection involved the nasopharynx and the larynx. The soreness behind the palate and the stuffiness in the ears were relieved and the duration of the condition shortened. After one treatment the injection in the pharynx was often much lessened and usually three or four treatments effected complete relief.

4. I now use 100,000 units of penicillin calcium and 100,000 units of streptomycin in propylene glycol, 1 to 2 cc.

Patients with acute follicular tonsillitis were occasionally given one inhalation on the day on which they first appeared at the office. They were then sent to bed, and the usual home treatment, sulfadiazine by mouth and gargles, was prescribed. In some cases penicillin (300,000 units) in oil and wax was injected into the buttocks at the same time; so no conclusions can be drawn as to the effect of penicillin aerosol in these cases. No patient with this type of disease was treated exclusively by inhalations.

One patient who, it turned out later, had a virus pneumonia was treated with daily inhalations of 100,000 units for four days. This particular patient, a man 28 years of age, did not show any favorable response to the inhalations but showed an irritation of the skin which the mask covered. The skin became red and sore, the lips moderately swollen, and the vestibules of the nares irritated. This apparently was an allergic response to the penicillin and disappeared a few days after the treatment was discontinued. He was sent to the hospital because of cough and a continuous low grade fever, and the internist stated that the patient had a virus pneumonia. Following this experience, if any patient with bronchial symptoms shows any fever after the first treatment, he is sent to an internist for a thorough check-up. Such patients may need more than office treatment. The purpose of this paper is to report on the practicability of office use of penicillin aerosol, and it is realized that all conditions cannot be handled in this manner.

All in all, the inhalations of penicillin have been a valuable adjunct in my office. It may seem from what has been said in the foregoing pages that all patients who come into my office are treated with penicillin aerosol. That really is not so; only those in whom the infection is difficult to reach by simple means or "gaggy" patients or those in whom the usual treatment is not successful are given penicillin by inhalation. This is the simplest method of introducing medicine into the laryngeal or tracheobronchial tree and is appreciated by the patient who has had to have his pharynx or larynx anesthetized in order that the lower respiratory tract might be treated. Many persons with low grade chronic sinusitis and a postnasal drip, in whose case the usual treatments are of little value, get better results with less discomfort by inhalations, although these, too, do not often result in complete cures. When used in addition to other therapeutic means, they are a valuable aid. Possibly some of the bronchiectatic patients would show better results if the treatments were continued indefinitely or were given several times a day, but this is not too easily done when the patients must work and can spend only a certain length of time in a physician's office. Those patients who need penicillin by inhalation several times a day, such as bronchiectatic patients who are to undergo lobectomy, should be hospitalized and an inhalation of 10,000 units in 1 cc. of salt

solution given every hour during the day. This decreases the amount of secretion in the bronchial tree and is a wonderful aid to the thoracic surgeon and the patient during the operation. However, the ambulatory bronchiectatic patient who is working gets much relief from a daily inhalation of 100,000 units. When possible, more than one treatment a day is given, and the patient can continue to work at his daily job.

When the inhalations of penicillin are not well tolerated, they are discontinued. One of the patients with bronchiectasis had an asthmatic attack during the night following an inhalation given the previous afternoon. This occurred on two successive nights. After each inhalation he felt fine, but twelve hours later the asthmatic attack developed. Since he had had similar asthmatic attacks occasionally before taking the inhalations of penicillin and did not have them when penicillin had been instilled into the tracheobronchial tree with a laryngeal syringe, I believe some other factor and not the penicillin may have been responsible for the attacks.[5]

Now that streptomycin and propylene glycol are available, the efficacy of inhalation therapy will be greatly enhanced. The rationale of inhalation therapy has been proved through the past many years with drugs of various kinds, and as long as one does not attempt the impossible, it should prove to be a valuable adjunct to the otolaryngologists' armamentarium. The oxygen itself may be of some therapeutic value and when combined with the proper antibiotic substance or drug may have a real synergistic effect.

The three case reports which follow are representative of many in my files. They do not show spectacular results, but in my estimation the patients were definitely helped by the inhalation treatments.

R. T., a man aged 39 years, complained of "sinus attacks" which had been occurring for the past seven years. The illness for which he sought treatment had been present about ten days and consisted of headache and nasal discharge. Pus was present in both nares, and the maxillary sinuses were dark on transillumination. The secretion was removed by suction, and 100,000 units of penicillin aerosol was given. Two days later the patient felt better, though the left naris was clogged. He was given another 100,000 units of penicillin by inhalation. When he returned two days later he had pain in his left cheek, and there was pus in

5. There have been several allergic reactions to the use of penicillin aerosol in salt solution. One patient (who had virus pneumonia) had a very mild reaction of his skin where the face mask had been. However, I have observed 2 cases of severe asthmatic reactions in which heroic measures had to be used to relieve the patient: "benadryl (beta dimethylaminoethyl benzohydryl ether hydrochloride), aminophyllin injection U. S. P., epinephrine inhalation U. S. P., oxygen, etc. Now if a patient shows any cutaneous reaction or even a slight wheezing following a treatment, the inhalations are discontinued until skin tests are made with the antibiotics. It has been recommended that "benadryl" be added to the mixture which is inhaled to prevent an allergic reaction, but I have had no experience yet with this combination.

the left middle meatus. The left maxillary sinus was irrigated, with recovery of a large amount of pus and mucopus. Three days later another irrigation of the left maxillary sinus revealed a small amount of mucopus. Within the next nine days penicillin aerosol was given six times, 100,000 units each time, and another inhalation ten days later. Four months after the last inhalation the patient stated that he had never had such long-lasting relief from his nasal symptoms. His nose, he said, felt clear, there was no discharge and there was no cough at night, although these symptoms had been present constantly for the seven years prior to treatment.

E. B., a 13 year old girl, according to her mother, had always had the sniffles. There was constant clogging of the nose. The ears felt stuffy and often painful, and the hearing was below normal. Some pains had developed in her fingers and several other joints, and occasionally she had an elevation of temperature of 1 degree or more. There was some tissue in the tonsillar fossae, but the child would not allow any cauterization, and the mother would not consent to any operation for the removal of this tissue. Culture of the nasal secretion showed Staphylococcus aureus, nonhemolytic. Roentgenograms of the sinuses showed diffuse chronic pansinusitis, worse in the right antrum. Allergy was not present. Fifty thousand units of penicillin aerosol was given daily for ten doses, and 100,000 units almost daily for six doses. The pains of the joints disappeared, the nares became open, the stuffy feeling in the ears was relieved and the hearing improved. Children of the type of this child object strenuously to all treatments of nose or throat, and consequently little is done for them. This child liked the penicillin inhalations and therefore had no fear of returning for enough inhalations to be of value to her. In more cooperative children equally good results might be obtained with many other kinds of treatment, but for those children who do not cooperate and whose conditions are amenable to penicillin inhalations, one has in these inhalations a valuable form of therapy.

Mrs. D. B., aged 44 years, complained of cough and sore throat of three days' duration and of nasal discharge of many months' duration. The mucosa of the nares was red and boggy, the sinuses were clear on transillumination, the tonsils were small, and there was inflamed lymphoid tissue on the posterior wall of the pharynx and the lateral bands. She was given six inhalations of penicillin, one daily, with complete relief of the cough and the sore throat; but what pleased her most was the elimination of the nasal discharge which had annoyed her so greatly. Three months later she was still relieved, and there was yet no return of the nasal discharge.

CONCLUSIONS

1. Penicillin by inhalation is of value in the treatment of many conditions of the upper and lower respiratory tract, such as acute sinusitis, nasopharyngitis, pharyngitis, laryngitis, tracheobronchitis and infectious asthma.

2. From 50,000 to 100,000 units of penicillin in 1 to 1.5 cc. of salt solution inhaled daily from one to ten days will usually suffice to cure or to relieve most simple infections of the respiratory tract.

3. The results are best in conditions due to organisms affected by penicillin.

4. The treatments can be given by the office nurse. Thus the doctor is relieved of seeing the patient except when a check-up is necessary.

5. Penicillin aerosol is not a cure-all and does not relieve the doctor of giving his usual treatments in most cases, but is a valuable adjunct in some cases and the sole therapy in others.

6. (This conclusion is added just before publication.) Penicillin aerosol in salt solution is excellent for infections of the respiratory tract, but even better results have been observed since propylene glycol was substituted for the salt solution. A combination of penicillin calcium, 100,000 units, and streptomycin, 100,000 units, in propylene glycol, 1 to 2 cc., is now used and is found to be superior to penicillin alone.

Mrs. Virginia Renner as office nurse, by her enthusiasm, furthered the use of penicillin aerosol in my office.

1835 Eye Street, N. W.

THE TONSIL PROBLEM

RICHARD WALDAPFEL, M.D.
GRAND JUNCTION, COLO.

SOME may be surprised that such a problem exists. Many times every day the otolaryngologist examines tonsils. They have been examined under normal and under pathologic conditions for more than a thousand years. They have been removed innumerable times for almost a century, and if anything should be clear, it should be the pathology of these organs.

This is not the case. There are secrets about them no matter from what angle one looks at them—clinical, anatomic, physiologic or pathologic-anatomic. I believe these secrets to be a subject especially worthy of extensive research. Furthermore, I have in mind to stimulate clinical interest in the pathology of the lymphatic tissues, which future investigations may well prove to be among the most essential structures of the body.

I shall start with the clinical side of the problem. The acute infection of the tonsils is called "tonsillitis" (in European countries, "angina," a Greek name which means a throat condition accompanied by signs of obstruction). Every one knows the picture: both tonsils increased in size, with numerous yellow patches becoming visible, corresponding to the openings of the crypts and containing streptococci. The patient is seriously ill and has a high fever. The symptoms subside in most cases and under average conditions in three to five days.

The disease is very simply described, indeed, but not so simply explained. For its course has no analogy in any other streptococcic infection. A streptococcic infection of the index finger, for example, will gather, come to a head, break or be absorbed, and that will take a certain time, which varies in every case. If it spreads, it will affect a lymph gland, perhaps one in the elbow, perhaps one in the axilla. It will never affect a finger of the other hand, let alone appear there simultaneously. But the tonsillar infection is bilateral; it starts and ends in both tonsils at the same time. This disease is cyclic; that means it runs a certain cycle, has a certain duration, terminates in the majority of cases in a certain time—like an acute infectious disease, such as measles, small-

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pox or scarlet fever. The disease affects not only the tonsils but often other parts of the lymphatic tissue of the pharynx, also. If it does, it remains strictly limited to this type of tissue and never oversteps the boundaries.

I could continue for some time describing mysterious unexplained details. Any one who has experienced tonsillitis following a nasal operation or packing of the nose must have been puzzled by it. The nose may have been operated on or packed on one side only, but the tonsillitis is on both sides. This kind of tonsillitis has been called, ever since its first description by B. Fränkel [1] (1896), "traumatic tonsillitis." And there is another type of tonsillitis which occurs epidemically; several members of the family, all the children occupying a classroom, get tonsillitis.

Let these few examples of clinical variations suffice to demonstrate some of the problems one is up against in explaining tonsillitis from a clinical point of view.

Many theories have been advanced in the effort to explain acute tonsillitis. One of them, emanating from the first describer of traumatic tonsillitis, regards the tonsils as regional lymph glands of the nasal mucous membrane, which would explain tonsillitis following nasal infections very simply, like lymphadenitis. Another one anticipates a preliminary local preparation of the tonsils of some kind which activates the streptococci inhabiting the tonsils; by an unknown mechanism these bacteria, harmless under normal conditions, become virulent and dangerous enemies of the body. A third theory regards tonsillitis as a septic general disease with an unknown portal of infection. According to this theory, tonsillitis represents only the first local manifestation of the general infection.

I purposely omit many interesting theories and have selected only the principal ones to discuss briefly.

The conception that the tonsils are regional lymph glands of the nasal mucous membrane, connected with the latter by lymph vessels, cannot explain the simultaneous infection of both tonsils and of other parts of the lymphoid tissue of the pharynx, occurring even after unilateral infection of, or operation on, the nose; nor can it explain the epidemic incidence of acute tonsillitis. In addition, Schlemmer [2] has shown in fundamental investigations that there are no lymph vessels leading from the nose to the tonsils; colored material injected into the nose is carried to the cervical lymph glands and around the tonsils but not into them. Therefore, an infection of the tonsils induced from

1. Fränkel, B.: The Infectious Nature of Lacunar Tonsillitis, Arch. f. Laryng. u. Rhin. **4**:130, 1896.

2. Schlemmer, F.: Anatomical, Experimental and Clinical Studies of the Tonsil Problem, Monatschr. f. Ohrenh. **55**:279, 1921.

the nose by way of the lymph vessels cannot occur. Nevertheless, tonsillitis following nasal encroachments remains a fact. I have been able to demonstrate a connection between the nose and the tonsils other than the lymph ways in experimental studies [3] by injecting 40 per cent iodized poppyseed oil in men and dyes, bacteria and stained leukocytes in dogs. The material injected into the nasal mucous membrane appeared in the tonsils but only in cases in which this material had been injected into the turbinates. From places other than the turbinates the injected substances were carried exclusively to the cervical lymph glands. This material was transported from the nose into the tonsils by the veins (fig. 1) carrying the blood from the cavernous tissue of the turbinates and communicating with the venous plexus of the pharynx including the tonsils. The material injected in one turbinate appeared in both tonsils,

Fig. 1.—Iodized oil injected into one nasal turbinate appears within the veins of both tonsils. This section through a small tonsillar artery and vein shows: *p*, parenchyma of the tonsil; *a*, artery; *v*, vein; *i o*, iodized oil within the lumen of the vein. (Black and white print of a "kodachrome" photomicrograph.)

a fact which is easily explained, since the veins of both sides of the pharynx communicate with each other. These studies, therefore, show without a doubt that a nasally induced infection of both tonsils is feasible.

The opinion that a preliminary local factor stimulates the streptococci in the tonsils to become virulent and to cause tonsillitis is based on the experience that changes in temperature, wetting of the skin and certain seasonal and atmospheric conditions are favorable to the occurrence of tonsillitis in many people. Nothing definite has been proved in this matter.

3. Waldapfel, R.: Experimental Histological Studies of the Tonsil Problem, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:400, 1933.

The theory that tonsillitis is merely a secondary local manifestation of a general infection with the portal of infection elsewhere in the body, outside of the tonsils, was promoted by Fein [4] (1921). According to this theory, there is no isolated acute infection of the tonsils alone, but always the entire lymphatic system is affected simultaneously and along with it both tonsils. In order to emphasize the general character of the disease, Fein proposed the name "anginosis" instead of "angina" or "tonsillitis," the latter of these names designating a local infection of the tonsils only. According to this theory, such conditions as rheumatism and nephritis are, like tonsillitis, equivalent coordinated manifestations of the same general disease and not the after-effects of tonsillitis. The same is true of appendicitis and pathologic changes occurring in other sections of the lymphatic system, which are all equivalent manifestations of the same general disease.

This theory has much in its favor. It would account for the cyclic character of tonsillitis, the simultaneous appearance of inflammation in both tonsils and in other parts of the lymphatic tissue of the pharynx, and the epidemic occurrence. There are some newer clinical and experimental findings, too, which are in its favor. I have recently published cases [5] in which there was simultaneous infection of the lymphatic tissue of the pharynx and of the conjunctiva (fig. 2) and which support Fein's theory that tonsillitis is merely a part of a general infection of the lymphatic system rather than a localized infection of the tonsils. To the same conclusion point important observations made after vaccination, which I will mention later.

All these theories concerning the clinical aspect of the problem show that a wide field has been left open for investigation and the contribution of more knowledge.

No less puzzling are the secrets which surround the anatomic and the physiologic sides of the problem.

The palatine tonsils represent a part of the lymphatic "faucial ring" first described by Waldeyer [6]; the rest of the ring is formed by the lingual and the pharyngeal tonsil and by smaller aggregations of lymphatic tissue around the eustachian tubes, in the posterior pharyngeal wall, in the piriform sinuses and in the larynx. They all represent outposts of the lymphatic tissue, far advanced under the epithelium.

The microscopic structure of the lymphatic tissue in man and animals is everywhere the same. It consists of two distinct anatomic elements: parenchyma and secondary nodules (fig. 3 *A*). The latter, characteristic of, and found exclusively in, lymphatic tissue, are round or oval forma-

4. Fein, J.: Die Anginose, Vienna, Urban & Schwarzenberg, 1921.

5. Waldapfel, R.: Infection of Lymphoid Tissue of the Pharynx and of the Conjunctiva, Arch. Ophth. **31**:331 (April) 1944.

6. Waldeyer, W.: The Lymphatic Faucial Ring, Deutsche med. Wchnschr., 1884.

tions and consist of a light center and a dark marginal zone. The light center contains numerous cells with a large amount of protoplasm which stain lighter and make the centers stand out as lighter foci in the parenchyma. These cells are of an exquisite phagocytic nature and are called macrophages. They contain dark particles, first observed by Flemming[7] (1885) and interpreted as products of cell metabolism (fig. 3 *B*). Not knowing their nature, he called them simply "tingible corpuscles." The marginal zone of the secondary nodules consists of densely packed lymphocytes and appears, therefore, in the slides as a dark ring.

What distinguishes the lymphatic tissue of Waldeyer's ring from the lymphatic tissue of the other areas is its relation to the epithelium. In Waldeyer's ring the epithelium of the pharynx comes into direct contact with the lymphatic tissue and an extensive surface of contact between

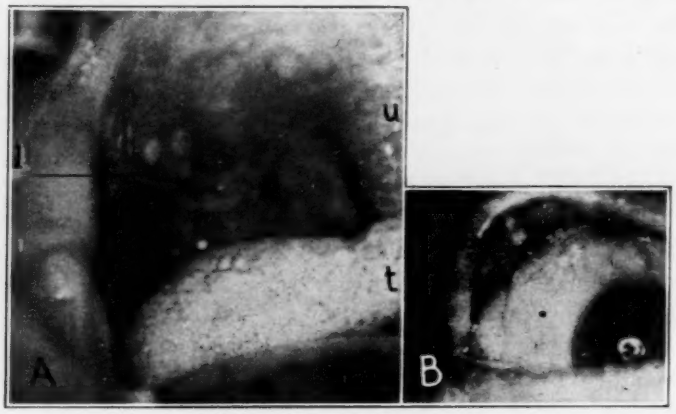

Fig. 2.—Simultaneous infection of the lymphatic tissue of the pharynx and of the conjunctiva of the same patient. *A* illustrates the condition of the right side of the pharynx. In a tonsil tag (*l*) two light patches are visible which represent subepithelial abscesses in secondary nodules of the lymphatic tissue. *U* designates the uvula; *t*, the tongue.

B illustrates the condition of the conjunctiva of the upper lid of the right eye. Within an inflamed area two light patches are discernible which represent a pathologic involvement analogous to the pharyngeal condition. (Black and white prints of "kodachrome" transparencies.)

these two structures is established, a factor which without doubt plays an important part in the physiology of the tonsils.

The simplest type of this junction is represented in the rabbit's tonsil (fig. 4). It is a single crypt formed by a sinking in of the pharyngeal epithelium and surrounded by a layer of lymphatic tissue in which one can easily distinguish the dark parenchyma and the lighter secondary

7. Flemming, W.: Studies of Tissue Regeneration, Arch. f. mikr. Anat. **24**:50, 1885.

nodules. This is the basic unit of every tonsil, and the human tonsil is built up from many such units.

The main secret of the lymphatic tissue centers around the secondary nodules. Flemming, who first described them, in 1885, concluded on the basis of numerous mitotic figures in them that they are main production sites for the formation of lymphocytes of the blood, which originate in the light center of the secondary nodules and are deposited in the dark marginal zone, which consists of newly formed lymphocytes. On

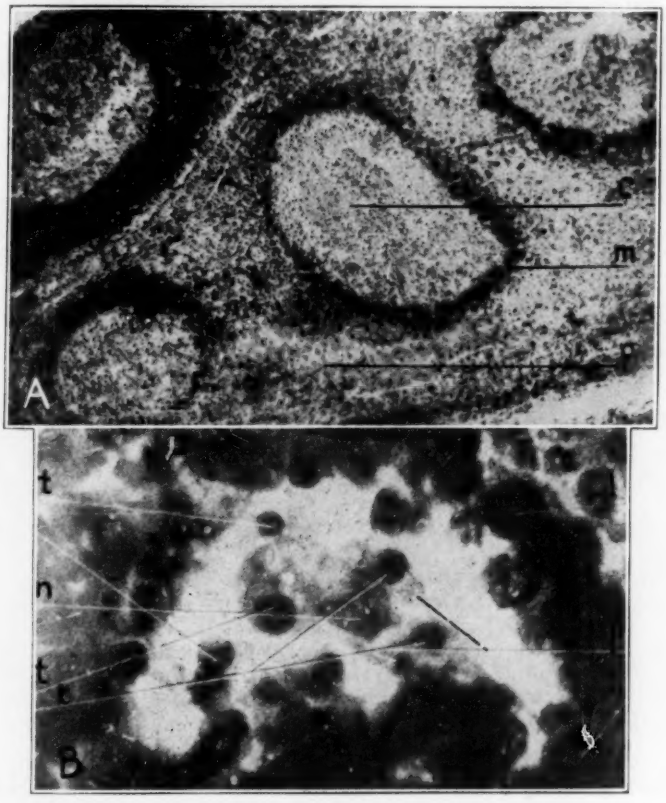

Fig. 3.—*A* shows constituents of the lymphatic tissue: *p*, parenchyma; *c*, the light center of a secondary nodule; *m*, the dark marginal zone of a secondary nodule. Four secondary nodules are seen. In *B* is a large phagocytic cell (macrophage) from the light center of a secondary nodule, greatly magnified. Within its protoplasm (*p*) a large nucleus can be seen (*n*), also four dark particles (*t*) of various sizes, which are Flemming's "tingible corpuscles." *L* indicates lymphocytes.

account of this hemopoietic function he named these secondary nodules "germinative centers."

Later observers doubted the rightness of this interpretation, laid the lymphocytic production to the parenchyma and attributed to the secon-

dary nodules primarily some other function. Hellman [8] and his school have regarded them as "reaction or defense centers" against toxic and bacterial irritants of low intensity continually invading the lymphatic tissue. The numerous mitotic figures on which Flemming built up his theory of the production of new lymphocytes may as well be explained as a reactive process following the invasion of toxic substances. Further reasons for this argumentation are: The "tingible corpuscles" could not be explained by Flemming himself. They are easily explainable as remainders of destroyed cells and nuclear fragments and can even be expected there if the secondary nodules are reactive centers. Furthermore, secondary nodules appear only several months after birth; in the fetus and the newborn they do not occur. The formation of lymphocytes

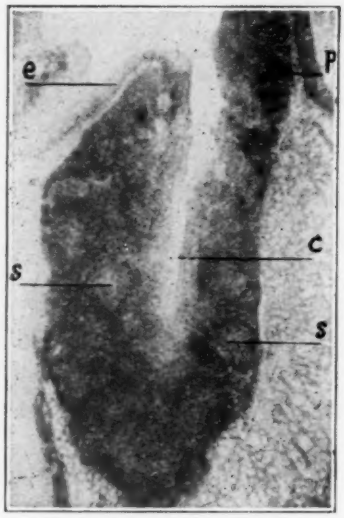

Fig. 4.—Section through a rabbit's tonsil. Note that pharyngeal epithelium (*e*) sinks in to form one crypt (*c*) surrounded by a layer of lymphatic tissue containing the typical lymphatic elements, parenchyma (*p*) and secondary nodules (*s*).

starts during embryonic life and is extensive during the last fetal months and the first postnatal months. That means lymphocytes appear in the body long before secondary nodules appear; so the latter cannot be the production centers of the former. Furthermore, bacteria injected into rabbits provoke extensive formation of secondary nodules.[9] Finally,

8. Hellman, T.: The Significance of the Secondary Nodules, Beitr. z. path. Anat. u. z. allg. Path. **68**:333, 1921.

9. Hellman, T.: The Lymphatic Faucial Ring, in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1927, vol. 5, pt. 1, pp. 245-289. Ehrich, W.: III. Studies of the Lymphatic Tissue. J. Exper. Med. **49**:347, 1929.

some crucial experiments: Guinea pigs reared completely sterile and free from bacteria in ingeniously constructed apparatus from birth to 2 months of age—that is, to the age in which secondary nodules are well formed in the normal guinea pig—do not show secondary nodules,[10] and rabbits infected regularly with a daily injection of bacteria from birth to the age of 3 weeks—that is, to the age in which secondary nodules are not yet formed in the normal rabbit—show secondary nodules prematurely, in about half the time of the normal development.[11]

All this evidence is strongly in favor of Hellman's defensive theory and against Flemming's hemopoietic theory.

In recent years a new and important angle of the problem has been stressed, namely, that of the part which the secondary nodules play in the production of antibodies. The secondary nodules become much enlarged and increased in number during the process of immunization, and there is sufficient evidence to support the belief that the secondary nodules have a definite essential function associated with immunization.[12] In the alimentary and respiratory tracts they are separated from the outside world by a single epithelial layer only, and exposure to bacteria is perhaps constant. Here a continuous "autovaccination" of the body may take place.

One cannot conclude the chapter of the protective function of the lymphatic tissue and of the tonsils without touching on the question of how substances are eliminated from them. There has been a great variety of opinions as to whether there is a "centrifugal" movement toward the pharynx and, if so, whether there is an active or a passive movement. I do not want to go into too many details but merely want to mention that there is microscopic evidence [8] that this route from the tonsils through the epithelium into the pharynx exists (fig. 5). One of the latest developments in the field of biologic function of the tonsils is the demonstration that vaccine virus is eliminated through the tonsils. Gins and co-workers,[13] inspired by observations made in vaccinated children by the pediatrician Orgler,[14] who saw tonsillitis occurring after vaccination, succeeded in demonstrating the vaccine virus on the

10. Glimstedt, G.: Life Without Bacteria, Anat. Anz., 1932.

11. Waldapfel, R.: The Germinal Center Question, Wien. klin. Wchnschr. **40**:728, 1927.

12. Hellman, T., and White, G.: The Lymphatic Tissue During an Immunization Process, Virchows Arch. f. path. Anat. **278**:221, 1930. Kaiser, A. D.: Results of Tonsillectomy, J. A. M. A. **95**:837 (Sept. 20) 1930. Miller, R. E.: The Secondary Nodules of Lymphatic Tissue, New England J. Med. **209**:591, 1933.

13. Gins, H. A.: The Problem of the Biologic Function of the Tonsils, Fortschr. d. Zahnh. **6**:281, 1930. Gins, H. A.; Hackenthal, H., and Kamentzewa, N.: Experimental Investigations of the Generalization of the Vaccine Virus in Man and Guinea Pig, Ztschr. f. Hyg. u. Infektionskr. **110**:429, 1929.

14. Orgler, A.: On Accompanying Tonsillitis, Jahrb. f. Kinderh. **99**:243, 1922.

tonsils and the mucosa of the upper respiratory tract in man and in the experimental animal three to five days after preceding cutaneous vaccination. The same was true with the appendix.[15] Both reports are important evidence that an elimination mechanism exists in the tonsils and in the subepithelial lymphatic tissue of the alimentary and the respiratory tract, and are certainly going to play an important role in the interpretation of the genesis of acute tonsillitis.

Finally, a few words about the pathologic-anatomic picture of acute tonsillitis. What is it in fact that one calls acute or follicular or lacunar tonsillitis? The textbooks do not say much. The pathologic anatomist hardly ever sees acute tonsillitis, and until not too long ago one was in the dark about the most primitive questions. One had a simple conception of it, taking for granted that pus fills every crypt like a plug and

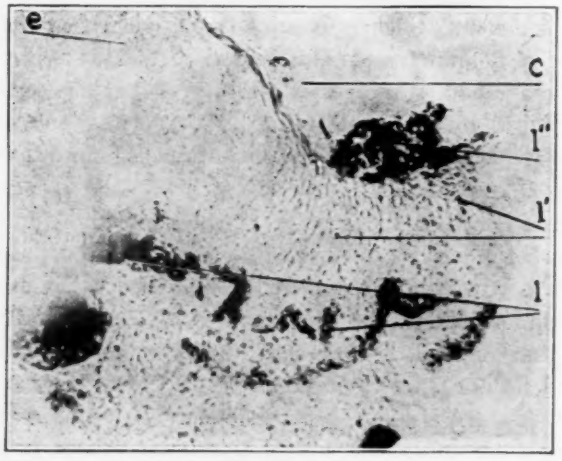

Fig. 5.—Tonsillar elimination. Stained leukocytes injected into the blood stream of a dog appear on the pharyngeal surface of the tonsillar epithelium after crossing the epithelium from the subepithelial blood vessels. An *e* indicates epithelium; *l*, subepithelial arterial capillaries filled with masses of stained leukocytes; *l'*, stained leukocytes crossing the epithelium; *l''*, stained leukocytes after passing the epithelium collecting in a crypt (*c*). This is a photograph of an unstained microscopic slide; the leukocytes were stained with congo red before injection.

that at the opening of the crypt the pus extends to the surface of the tonsil and thus produces the clinical picture every one knows. This conception of an infection limited to the surface epithelium and filling the crypts with a leukocytic exudate would indeed not harmonize with the severe clinical symptoms, the high fever and the serious abscess-forming complications occasionally following tonsillitis. A coincidence helped in obtaining the real pathologic-anatomic picture. In a case of acute tonsil-

15. Roth, P.: Studies on the Vaccine Virus Elimination through the Lymphatic Organs of the Intestine, Ztschr. f. Hyg. u. Infektionskr. **122**:159, 1939.

litis of a child, with tonsils so immensely swollen that they caused suffocation and a tracheotomy would have become necessary, the tonsils were removed and microscopically studied.[16] The study showed: Acute tonsillitis is not a superficial infection; it involves the tonsil as a whole and is a deep-reaching phlegmonous infection with multiple abscess formation and destruction of large parts of the parenchyma (fig. 6). What appears as a "patch" is not a mere cast of exudate in a crypt but quite as often an abscess perforating from inside the parenchyma to the surface of the tonsil.

The secondary nodules, during acute tonsillitis, are also the seat of important changes (fig. 7 *A* and *B*). In their centers develop circumscribed small abscesses with streptococci which apparently do not communicate with the exudate of the crypts. In later stages[17] the streptococci disappear, and what is left of the abscesses within the

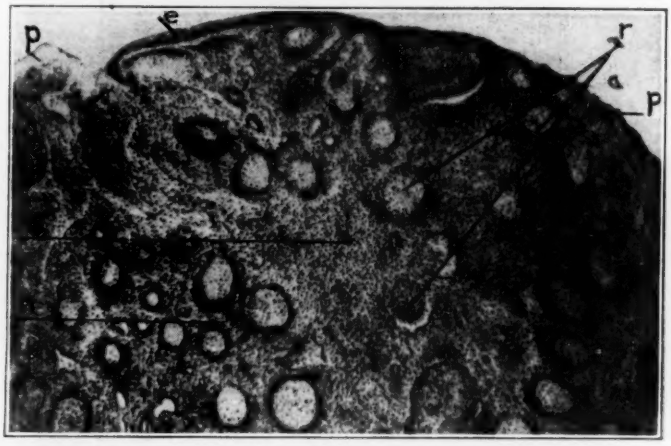

Fig. 6.—Destruction of large parts of the tonsillar tissue in acute tonsillitis. At the top of the section *e* indicates epithelium of the tonsil; *n*, a fairly normal section of tonsillar tissue with regular parenchyma and secondary nodules; *a*, an abscess destroying the normal tonsillar structure, containing pus and streptococci and perforating through a crypt to the surface of the tonsil (*p*), appearing here as a yellow patch; *r*, remainders of secondary nodules within the abscess.

involved secondary nodules are areas of fatty degeneration centrally located and indicating previous cell destruction and degeneration in these places (fig. 7 *C*)—a "defense job" accomplished by the secondary nodules. These pathologic-anatomic findings explain the symptoms

16. Grossman, B., and Waldapfel, R.: Lacunar Tonsillitis, Acta oto-laryng., 1925, vol. 10.

17. Waldapfel, R.: Further Investigations of the Changes in the Lymph Follicles in Lacunar Tonsillitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:217, 1926.

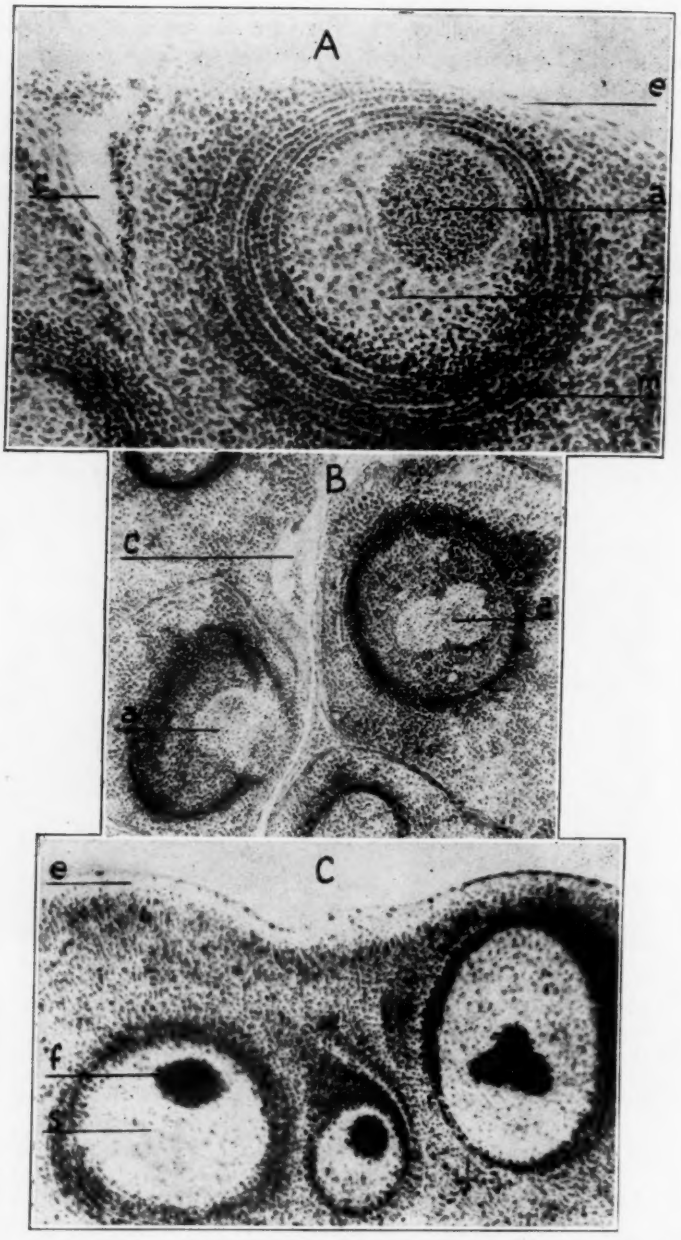

Fig. 7.—*A*, secondary nodule as it appears during acute tonsillitis. Within the light center of the secondary nodule (*z*) a sharply limited small abscess (*a*) has developed which shows no communication with the exudate of the crypt (*c*). The abscess contains a pure culture of streptococci. An *e* indicates the epithelium of the tonsil; *m*, the dark marginal zone of the secondary nodule.

B, multiple abscesses (*a*) like that in *A* within secondary nodules along a crypt (*c*), slightly different in shape and location but not directly connected with the exudate of the crypt. Pictures like this suggest a hematogenous infection of the secondary nodules.

C, areas of fatty degeneration within secondary nodules in a tonsil recovering from tonsillitis. They are temporary remainders of bacterial and cellular destruction occurring within the secondary nodules during the acute stage. Finally these areas are eliminated from the secondary nodules, and a few days after the tonsillitis has cleared up the normal picture of the secondary nodules is found throughout.

much more satisfactorily than the previous conception did. A patient with acute tonsillitis is, therefore, on the grounds of these findings alone, a seriously ill patient and requires the most careful attention.

The central abscesses within the secondary nodules and without direct connection with the exudate of the crypts strongly suggest a hematogenous infection of the secondary nodules; if this is correct, it represents another proof that tonsillitis not only is not a superficial infection of the tonsils but very likely is even a temporary septicemia caused by streptococci.

I have tried to show how much is known about the tonsils and at the same time how little. I have not even attempted to be complete, as that would have been far beyond the scope of this presentation. As one grows older and more experienced, one becomes more doubtful about many facts which one accepted automatically and considered beyond doubt in younger days. This is true also of the tonsil question. By remembering in one's daily routine work a few of the details pointed out here, one will perhaps find it less automatic and more interesting.

NASAL SENSITIVITY TO VASOCONSTRICTORS

R. WESLEY WRIGHT, M.D.
PALO ALTO, CALIF.

MEDICAL literature contains many examples of pathologic conditions arising from the use of drugs given for the relief of various diseases or symptoms. One of the earliest examples is that represented in the symptoms due to withdrawal of cocaine, a drug which was not at first known to be habit forming. Many doctors themselves became addicted to its use. Two striking examples in recent years of pathologic states arising from the action of drugs are cataract formation from the use of dinitrophenol and granulocytopenia from the use of aminopyrine. Aminopyrine had come into wide use several years prior to the reports of a strange new disease, termed "agranulocytosis." A causal connection with the drug was not discovered until some years later. Dinitrophenol was used for reducing weight for some time before it was recognized to be a cause of cataracts. In medical literature there are many other instances of the same nature.

Nasal sensitivity to vasoconstrictors is not of so serious a nature as the examples cited but may produce great discomfort over a long period.

With the widespread use of nasal drops, which can be obtained with little restriction, I believe that there will be an increasing number of patients suffering from nasal symptoms which often will be unrecognized as those of drug sensitization.

Kully,[1] in an excellent article, stated that there are at least 240 nasal vasoconstrictor compounds in various forms. He reviews the pharmacology of the adrenergic or sympathomimetic drugs, the class to which the vasoconstrictors belong.

The symptom which is predominant as the presenting complaint is nasal stuffiness. In cases in which sensitivity has been established, the history is typical. The patient comes complaining of nasal congestion which is constantly present. Nasal drops, whichever are being used, will open the nasal passages well, and the breathing space then seems normal, but the congestion returns in about three hours. This approximate period is almost always constant. If this condition is recognized by the rhinologist as a sensitivity he will ask to see the "drops" used, and the patient will produce them from a pocket or handbag. The dependence on

1. Kully, B. M.: The Use and Abuse of Nasal Vasoconstrictor Medications, J. A. M. A. **127**:307 (Feb. 10) 1945.

the medication is such that the medicament is carried at all times by the patient. There is usually no complaint of nasal or postnasal discharge by the time the patient is seen for this condition. That has passed. Typically the nose is dry but it may be moist. There is one other symptom of a more serious nature which is occasionally complained of, but is elicited only on questioning. This is loss of the sense of smell. Several patients have complained that complete anosmia occurs during the condition for which the drops are used. Any definite opinion as to a causal relationship between the anosmia and the nasal medication is not warranted at this time, but certainly the possibility deserves consideration. In some cases the anosmia noted has not cleared up with the clearing of the nasal congestion. There may be actual degeneration of the olfactory filaments. From the point of view of symptomatology, then, there is the single complaint of nasal stuffiness. With this congestion there are the attendant irritability, the worry over the condition and the annoyance of it.

The history of the onset of the condition for which the nasal medicament was used varies. Usually the condition was an acute cold or a recurrent attack of chronic sinusitis. In other instances the onset was the nasal congestion associated with pregnancy. Mohun [2] cited examples of nasal congestion arising during pregnancy. As this condition is now well recognized the patient is usually assured that the congestion will clear after delivery. In the cases which I shall report in the following pages, the patients were given drops to use until term, but the nasal congestion persisted. During colds they were given drops to use, or obtained them, and though the discharge usually ceased, the patients assumed that the condition had become sinusitis or some other chronic nasal condition, in which belief they were often fortified by a rhinologist. The longest history of nasal congestion due to medication which I have recorded was four years (case 3), during which there was the constant typical nasal stuffiness described.

The etiology of the condition is interesting. All the patients with this complaint whom I have seen had used the synthetic vasoconstrictors and not the natural products. I do not believe I have seen any who had such congestion from ephedrine. This may be at variance with the experience of others. The sensitivity spoken of is not that of most allergic reactions. It seems to be a compensatory vasodilatation which takes place in some people who have become "sensitized." The medication will always produce vasoconstriction, with relief, while most allergens produce the opposite effect. There is a vasodilatation which develops in about three to four hours. This vasodilatation has been found to continue in well established cases for about twenty-four to

2. Mohun, M.: Incidence of Vasomotor Rhinitis During Pregnancy, Arch. Otolaryng. **37**:699 (May) 1943.

forty-eight hours, before the vessels return to normal. Usually the patient is so uncomfortable and has so little understanding of the condition that medication is continually used for the relief. Thus they are the cause of their own discomfort.

Kully [1] stated that the primary vascular effect is constrictive, the capillaries, arterioles and venous sinuses being involved. Nasal secretion is diminished. When the vasoconstriction is severe and prolonged, a reversal reaction in the form of vasodilatation occurs. He states that the secondary reaction may be more prolonged and severe than the primary one.

The synthetic compounds have been produced to give the longer action. The literature accompanying these vasoconstrictor agents stress the prolonged effect. From this standpoint more reaction would be expected from these synthetic substances, because of the more prolonged effect, than from ephedrine.

Kully further stated that on the secondary vasodilatation the deeper venous structures are more involved than the superficial ones, which accounts for blanching. There may be a period of blanching but in my experience when the sensitivity is established the membrane is congested, slightly reddened and somewhat dry. I agree with Kully that the newer vasoconstrictors are not free from the secondary congestive effect as claimed for them by the makers.

The treatment of the nasal congestion is discontinuance of the medication. The relief of the stuffiness usually occurs in about twenty-four to forty-eight hours after cessation of the medication. In order to provide relief in the meantime I usually give a 2 per cent solution of a cocaine salt in small amounts, with instructions to use it sparingly. Usually little is found necessary. One patient needed only three drops. The sense of smell, loss of which may be on the same etiologic basis, has not recovered in 2 patients to this time.

REPORT OF CASES

CASE 1.—In March of 1939 a young woman came complaining of an acute cold. There were nasal stuffiness and discharge, with some general malaise as well. A synthetic vasoconstrictor was prescribed. Two months later she stated that she had had a succession of colds and that she was six months pregnant. The attacks seemed to be ordinary coryza. Nasal drops were being used. Later she stated that in the ten months after the first visit nasal stuffiness had developed, for which she had to use a strong vasoconstrictor every three hours. She was sent to the allergist, who found nothing on intradermal testing. She was then given potassium iodide, which seemed to help for a short time. The drops were still in use. She next went to the desert, which brought no relief. One year later, in April 1940, she complained of a cold as well as of stuffiness, which had been constant. In September 1940 the following summary was written. This was about eighteen months after the onset of the original coryza for which the nasal medication was given. "There has been nasal congestion since May of 1939 when

she was six months pregnant. It seemed to come on with a series of colds which may, however, have been coincidental. It was thought that the congestion would clear with the termination of the pregnancy but it has continued without change. There is a stuffiness which can be relieved only by a strong vasoconstrictor, and this is effective for about three hours. Her nose seems normal in the period of vasoconstriction, there being no discharge either nasal or postnasal. There are no symptoms attributable to sinusitis other than the intermittent nasal stuffiness. There have been many examinations. Two roentgen examinations of the sinuses have been made at an interval of months with nothing pathologic being found. Frequent nasal examinations have revealed only congestion of the inferior turbinates, which shrink readily. Displacements do no good whatever, and no discharge is obtained. Allergenic tests have revealed no positive reactions. She has taken potassium iodide for some months, with no relief. She has had an endocrinologic review with no positive findings. Cultures for bacteria that might cause sensitivity are negative. The conclusion is now reached that she is suffering from vasoconstrictor medication. She has been advised to give up all previously used drugs and to substitute a 2 per cent solution of cocaine hydrochloride, but to use it only if the congestion becomes extreme." Several days after the discontinuance of the nasal medication she was able to go through the night with no medication and no discomfort. She has had no trouble since this time.

Comment: This is the first case of vasoconstrictor sensitivity which came to my notice. It is obvious from the history that it was not recognized as such for many months.

Case 2.—A woman of about 35 years came with the complaint of nasal congestion of eighteen months' duration. The stuffiness came on without a cold or other infection of the respiratory tract in the second month of pregnancy. She was given a synthetic vasoconstrictor by her obstetrician and was assured that the condition would be relieved with the termination of pregnancy. There was no relief after delivery, and the medication was continued for relief. The drops were used every three to four hours. Definite relief of the stuffiness followed the medication. She felt that it was necessary to carry a small amount of "spray" with her at all times. There was slight postnasal discharge. She was advised by one rhinologist that the congestion had been present so long that there was no possibility of a cure. Another rhinologist advised that coagulation of the inferior turbinates would be necessary.

The nasal mucosa showed only a little reddening, congestion and dryness. There was a slightly glazed, dry appearance of the pharynx. The examination gave otherwise entirely negative results. With shrinking, the nasal membranes appeared essentially normal. No nasal or postnasal discharge was seen. The treatment consisted in complete avoidance of the nasal drops used. For relief she was given a 1 per cent solution of cocaine hydrochloride to be used only when the stuffiness was most uncomfortable. She reported in one week that only three drops of the solution were necessary, that the nasal congestion was relieved in about twenty-four hours and that there had been none since. There was no loss of olfaction at any time.

Case 3.—A young woman came with the complaint of nasal congestion of four years' duration. Examination showed a dry, slightly reddened, congested nasal mucosa, which allowed only slight breathing space. When the tissues were shrunken by vasoconstrictors, which could be accomplished readily, the nasal membrane and space appeared normal. No discharge was seen nasally or postnasally.

The history brought out the fact that the nasal congestion came on four years previously when there was an acute infection of the respiratory tract, for which nasal drops were used. A synthetic vasoconstrictor was employed for relief, which opened her nose well at all times. Gradually the discharge ceased, but there was continued congestion. It was necessary to use the drops about every four hours, and for this reason she always carried the medicament with her. She had been seen several times by rhinologists who treated her locally, without relief; she was advised to continue the use of a vasoconstrictor for relief. This patient's treatment was discontinuance of nasal medication. After two days she was completely relieved of the nasal congestion and has remained so. There was no anosmia.

CASE 4.—A middle-aged man was seen, who complained that he had suffered from nasal stuffiness for four years. The onset of the congestion occurred with a cold. The discharge of the cold cleared, but the congestion remained. There was also a complaint of complete loss of the sense of smell. During the cold he obtained relief from the congestion with a synthetic vasoconstrictor, which he continued to use, mostly at night, for the four years following. There was always prompt relief from the medication. The loss of the sense of smell came on after two years of nasal congestion and medication. Examination revealed a congested nasal mucosa, which was dry in appearance. There was some polypoid degeneration in each middle meatus. Vasoconstrictor nasal sensitivity was thought to be the cause of the stuffiness, and complete discontinuance of the use of the drug was advised. After a few nights there was no further stuffiness. Examination then revealed some pus in the pharynx and in the middle meatuses. Both antrums were found to contain pus. The discharge from this source he thought had been present many years. There was no return of the stuffiness, but to date, several weeks later, there is no relief of the anosmia.

CASE 5.—A man of 54 years was seen complaining of nasal stuffiness of twenty-five days' standing. There had been a submucous resection with some consequent nasal stuffiness. For the relief of this he had used 3 ounces (93 Gm.) of a synthetic vasoconstrictor in the twenty-five day period following the operation. There was good space after the shrinking of the tissues. There was no discharge at any time. He stated, however, that he thought the operation had been complicated by a slight cold, and therefore he had used the medication for relief. The relief continued for about three hours, and it was necessary for this reason to carry the "drops" with him at all times. Examination showed a slightly reddened and dry nasal mucosa. There was good space when the tissues were shrunken, and the septum had been well resected and had healed well. There was no discharge. A diagnosis of sensitivity to vasoconstrictor medication was made and a 2 per cent solution of cocaine hydrochloride was substituted, to be used only when the congestion became severe. The condition cleared up entirely within twenty-four hours.

SUMMARY

Vasoconstrictors are capable of producing a sensitivity which is manifested by nasal congestion. The vasoconstrictors most frequently found to be at fault are the synthetic ones. This may be because of the greater proportion of the synthetic medicament used, but also because of the more severe vasoconstriction. Relief is obtained promptly by discontinuance of the use of the drug. An additional symptom, anosmia, has been encountered, which may be on the same etiologic basis.

627 University Avenue.

MICROSCOPIC STUDY OF THE INNER EAR OF THE ATAXIC RABBIT

M. V. ANDERS, A.B., M.Sc.
CLEVELAND

IN 1942 Sawin [1] reported evidence that the loss of coordination occurring in a race of domestic rabbits is hereditary. The neuropathogenesis of this disorder proved to be an initial degeneration of the vestibular and cochlear nuclei, with resulting breakdown of the central pathways of these systems.[2] The consistent pattern of damage seen in the central nuclei of the auditory and vestibular tracts raised the question of what structural changes might result in their sensory end organs, i. e., the cochlea and the vestibule. The present report concerns the histologic study of a series of inner ears of 10 ataxic rabbits and 5 entirely unrelated normal controls of as nearly the same ages as possible.

MATERIAL AND METHODS

The rabbits were observed until the ataxia was well advanced. They were then anesthetized, and Susa-Heidenhain fixative [3] instilled into the auditory bullas and inner ears. Decalcification was done in 5 per cent trichloroacetic acid; after washing and dehydration, the two inner ears and the central nuclei of the medulla oblongata were embedded as a unit in celloidin (a concentrated pyrotylin). The celloidin sections were cut at 12 microns; some were stained with Delafield's hematoxylin-eosin, and others, with iron-hematoxylin. Serial sections of the entire internal ear were studied and compared with sections of normal controls. All rabbits, both normal and ataxic, showing evidences of aural infection were excluded from this study.

OBSERVATIONS

The ears of the ataxic rabbits were studied for possible changes of the bony structures, for degeneration of the ganglions and nerve fibers and for evidence of damage of the maculas and cristas of the vestibule and the semicircular canals.

A part of this work was done with the aid of a Crile Research Scholarship.

From the Department of Anatomy, Western Reserve University School of Medicine.

1. Sawin, P. B.; Anders, M. V., and Johnson, R. B.: Ataxia a Hereditary Nervous Disorder of the Rabbit, Proc. Nat. Acad. Sc. **28**:123-127 (April) 1942.

2. Anders, M. V.: The Histopathology of a New Type of Hereditary Loss of Coordination in Domestic Rabbit, Am. J. Anat. **76**:183-199 (March) 1945.

3. Lee, B.: The Microtomists' Vademecum, edited by J. B. Gatenby and T. S. Painter, ed. 10, Philadelphia, P. Blakiston's Son & Co., 1937, p. 50.

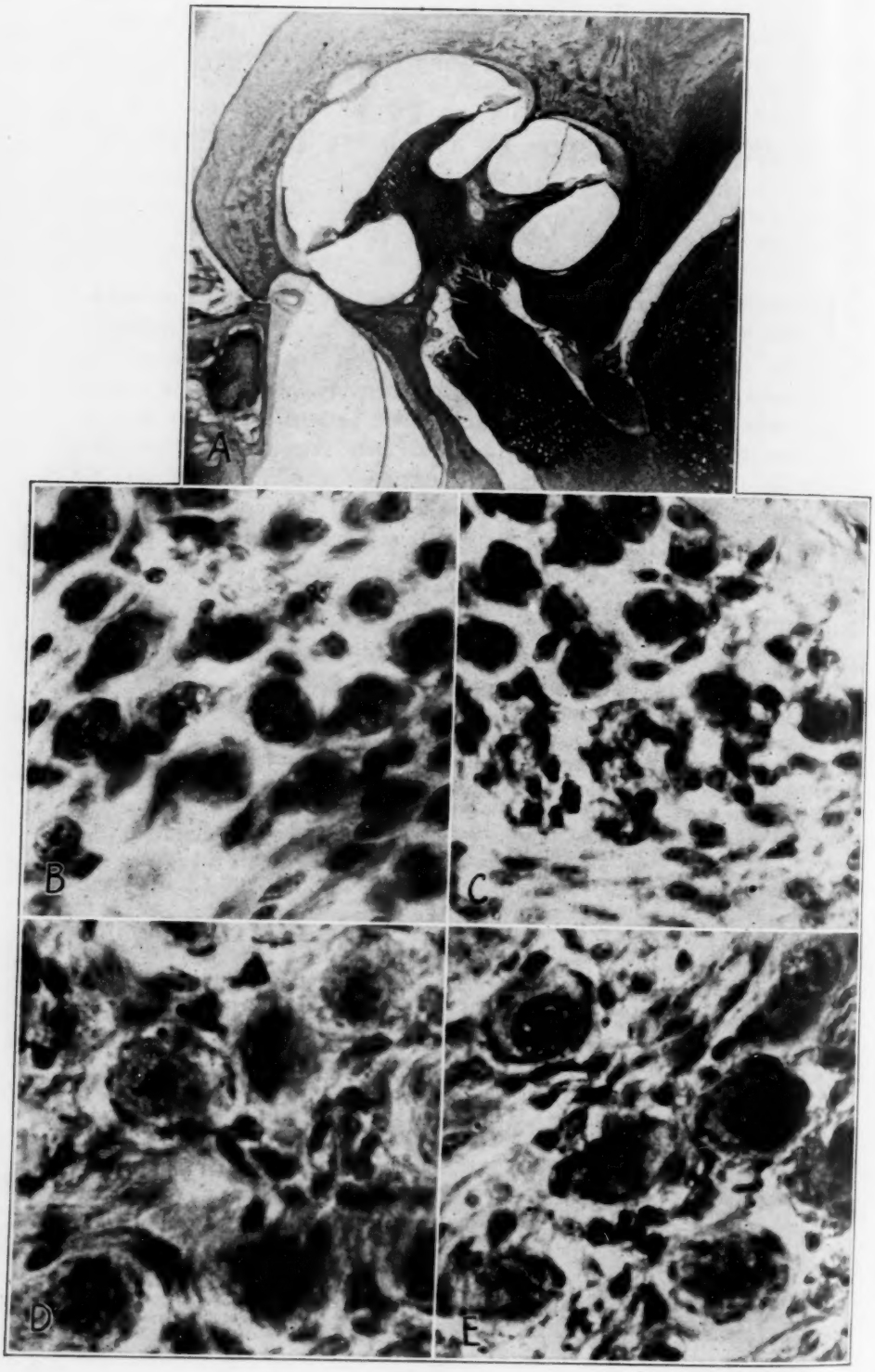

Figure 1

(See legend on opposite page)

No variations of structure could be found in the bone of the otic capsule. The osseous spiral laminas were well filled with cochlear nerve fibers, which showed no evidence of degeneration (fig. 1 *A*).

Both the spiral ganglion and the vestibular ganglion appeared to be composed of normal cells. No signs of damage could be found in either the cells or their respective fibers (fig. 1 *B* to *E*).

The stria vascularis and the organ of Corti compared well with those of normal controls. The only variations noted were changes of the position of the tectorial membrane and some occasional postmortem changes of the hair cells. Some of the control ears showed similar changes; the changes were considered, therefore, to be due to variations in the action and penetration of the fixative (fig. 2 *A* and *B*).

The hair cells and nerve fibers of the maculas and cristas of the vestibule all appeared to be normal (fig. 2 *C* and *D*).

The central nuclei of the vestibular and cochlear nerves showed the degenerative changes previously reported.[2] Figure 2 *E* illustrates the breakdown of the left medial vestibular nucleus of ataxic rabbit S936. The gray substance resembles a lacework of "holes" invaded by a myriad of "gitter" cells, with degenerating neurons and "shadow" cells in the center of the damage. Astrocytes and oligodendroglia cells are increased at the periphery.

It is clear from the foregoing observations that while there is consistent degeneration of the vestibular and cochlear nuclei in this family of rabbits, the damage does not progress to the peripheral ganglions and sensory end organs of the internal ear.

COMMENT

Tamari [4] reported atrophic degeneration of the cochlea present in a case of syphilitic gumma of the medulla oblongata. This was an infec-

4. Tamari, M.: Atrophic Degeneration of the Cochlea in Syphilitic Gumma of the Medulla Oblongata, Arch. Otolaryng. **34**:1014-1022 (Nov.) 1941.

Explanation of Figure 1

A, section through the modiolus and the eighth nerve of ataxic rabbit V120. Note the normal structures of cochlea and vestibule and the normal bony capsule. There is no loss of nerve fibers in the modiolus or the vestibular portion of the peripheral parts of the auditory nerve. Twelve micron celloidin section stained with Delafield's hematoxylin and eosin; × 15.

B, cells of the spiral ganglion of ataxic rabbit V120. All are normal appearing, with no evidence of distortion or loss. Normal nerve fibers are seen in the lower right hand corner. Twelve micron celloidin section stained with Delafield's hematoxylin and eosin; × 920.

C, cells of the spiral ganglion of control rabbit V101 for comparison with *B*. Nerve fibers may be seen in the lower part of the photograph. Twelve micron celloidin section stained with Delafield's hematoxylin and eosin; × 920.

D, normal-appearing cells of the vestibular ganglion of ataxic rabbit V120. No loss or distortion of the cells could be found. Twelve micron celloidin section stained with Delafield's hematoxylin and eosin; × 920.

E, cells of the vestibular ganglion of control rabbit V101 for comparison with *D*. Twelve micron celloidin section stained with Delafield's hematoxylin and eosin; × 920.

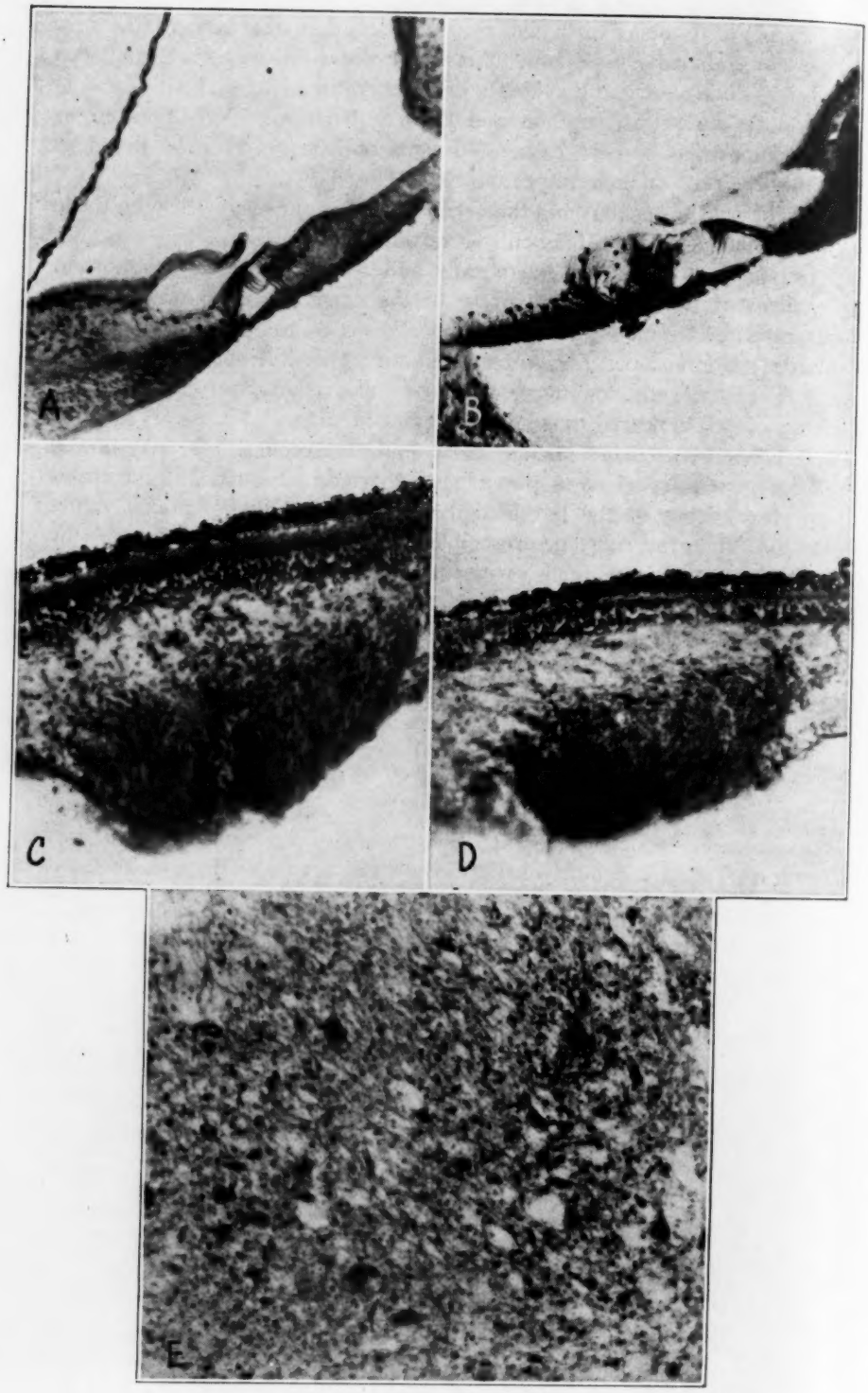

Figure 2

(See legend on opposite page)

tion of long standing, and the spirochetes had had ample time to invade the aural structures. Other aural infections are also recorded, in the various pathologic studies of the ear, as causes of damage of the inner ear. However, no reports concerning the aural structures of persons with hereditary nervous disorders resembling the type recorded herein have come to my attention.

Crowe [5] presented 2 cases of tumor of man which caused destruction of both branches of the eighth nerve close to the medulla oblongata. He stated that these cases indicate that the cochlear ganglion is an exception to the wallerian law of degeneration because a lesion of the cochlear nerve central to its ganglion causes secondary degeneration of the ganglion and the organ of Corti. However, a lesion central to the vestibular ganglion causes degeneration to that ganglion but not peripherally to it. He quoted the experimental work of Witmaack in 1911 on the auditory nerve in cats to support his findings.

It is clear that the work reported herein does not bear out Crowe's contention. However, the discrepancy might be due to a difference in the rabbit as contrasted to the human subject and the cat. Also, in the rabbit the period of time might not be sufficient to produce degeneration peripheral to the cochlear ganglion.

SUMMARY

The ears of 10 ataxic rabbits and 5 entirely unrelated rabbits used as normal controls, prepared with Susa-Heidenhain fixative, decalcified in 5 per cent trichloroacetic acid and embedded in celloidin, were examined for possible structural changes in the sensory end organs of the cochlea and vestibule.

5. Crowe, S. J.: Anatomic Changes in the Labyrinth Secondary to Cerebellopontile and Brain Tumors, Arch. Surg. **18**:982-991 (April) 1929.

Explanation of Figure 2

A, normal-appearing organ of Corti from the middle of the first turn of the cochlea of ataxic rabbit V120. Twelve micron celloidin section stained with Delafield's hematoxylin and eosin; × 150.

B, organ of Corti from the first turn of the cochlea of control rabbit V101. Reissner's membrane stained too faintly to show in the photograph. Twelve micron celloidin section stained with iron-hematoxylin; × 150.

C, macula from the vestibule of ataxic rabbit V120. Note the normal-appearing hair cells and nerve fibers. Twelve micron celloidin section stained with Delafield's hematoxylin and eosin; × 150.

D, macula from the vestibule of normal rabbit V101 for comparison with *C*. Twelve micron celloidin section stained with Delafield's hematoxylin and eosin; × 150.

E, left medial vestibular nucleus of ataxic rabbit S936 showing the lacework of "holes" consistently found in this hereditary degeneration. Note the damaged neurons and the myriads of tiny nuclei of the "gitter" cells in the network of degenerating fibers. Twelve micron celloidin section stained with Delafield's hematoxylin and eosin; × 150.

Since there appears to be no difference in the bone of the otic capsule, the cochlear and vestibular ganglions, the stria vascularis, the organ of Corti or the hair cells of the maculas and cristas of the vestibule between the normal and the ataxic animals, it is concluded that this single gene difference which has been shown to produce its principal defect in the cochlear and vestibular nuclei causes no detectable damage in their sensory end organs (cochlear and vestibular structures).

Dr. Paul B. Sawin, of Brown University, aided by a grant from the Rockefeller Foundation, supplied the rabbits afflicted with the hereditary loss of coordination.

Dr. M. H. Lurie gave technical advice.

MELKERSSON'S SYNDROME

Report of Five Cases, with Special Reference to the Pathologic Observations

KARSTEN KETTEL, M.D.
HILLERØD, DENMARK

BY MELKERSSON'S syndrome one understands (*a*) peripheral facial palsy, which may be familial and which may show a tendency to relapse, (*b*) angioneurotic facial edema and (*c*) lingua plicata.

Hübschmann [1] in 1894 and Rossolimo [2] in 1901 reported some cases in which besides relapsing facial palsy there was transient facial edema, while Melkersson [3] in 1928 was the first to establish the facies morbi of relapsing facial palsy and chronic edema of the face. Rosenthal [4] in 1931 added the third symptom, namely, lingua plicata. New and Kirch [5] in 1933 communicated the information that at the Mayo Clinic in the course of twenty-two years they had observed 67 patients who had chronic noninflammatory swelling of the lips and face and that 13 of these suffered from facial palsy.

In the Scandinavian medical literature the syndrome has been dealt with in detail by Ekbom and Wahlström [6] (1942), who have given a detailed statement of the question and have described five observations of their own, while two casuistic communications have been published from Denmark, one by Buch [7] (1943) and one by Scherwin [8] (1945). Five cases can now be published by me; the first case is identical with Buch's; nevertheless, I include it in the present communication partly because I want to make a few comments on it and partly because I desire to show the results of the treatment.

REPORT OF CASES

CASE 1 (figs. 1 and 2.)—A youth aged 18 years was admitted to the neurologic ward of the University Hospital in 1942. He had never had any symptoms of otitis media. At the ages of 2, 3 and 18 he had right-sided facial

1. Hübschmann, P.: Neurol. Centralbl. **13**:815, 1894.
2. Rossolimo, G. J.: Neurol. Centralbl. **20**:744, 1901.
3. Melkersson, E.: Hygiea **90**:737, 1928.
4. Rosenthal, C.: Ztschr. f. d. ges. Neurol. u. Psychiat. **131**:475, 1931.
5. New, G. B., and Kirch, W. A.: Permanent Enlargement of Lips and Face Secondary to Recurring Swellings and Associated with Facial Paralysis: Clinical Entity, J. A. M. A. **100**:1230 (April 22) 1933.
6. Ekbom, K. A., and Wahlström, A.: Nord. med. **15**:2373, 1942.
7. Buch, H.: Ugesk. f. læger **105**:1161, 1943.
8. Scherwin, J.: Ugesk. f. læger **107**:644, 1945.

palsy. After the age of about 16 there was constant swelling of the right upper lip, with periodic aggravations occurring without preceding symptoms, especially no local or regional disorders. On objective examination, definite right-sided peripheral facial palsy was observed, with Bell's phenomenon and difficulty in closing the eye. The movements of the mouth were also impaired on the left side, and the patient was unable to produce transverse wrinkles of the skin of the forehead. There was a firm pasty swelling of the entire right side of the upper lip extending well over the midline. The color of the skin was natural, the skin did not pit on pressure and there was no pulsation. Further, right-sided hyposmia and right-sided hypesthesia-algesia of the second trigeminal branch and the cornea were ascertained. The pressure of the spinal fluid was increased to 500 mm. of water, and Queckenstedt's experiment produced only a slight rise. The cell count was 0/3; the albumin content was 10 and the globulin content 1; the Pandy test was positive; the Wassermann test was negative.

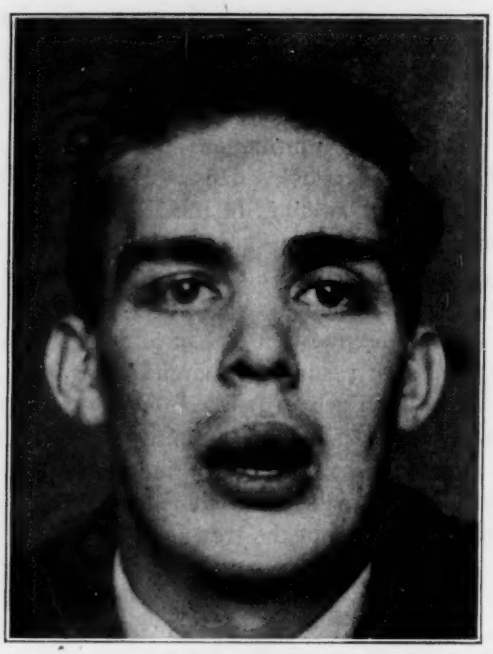

Fig. 1 (case 1).—Appearance of the patient, aged 18, when admitted to the hospital for treatment of a firm pasty swelling of the right side of the upper lip. Two years earlier he had shown in addition right-sided peripheral facial palsy with Bell's phenomenon and difficulty in closing the eye.

Buch suggested the possibility that in this case the causal condition might have been basal arachnoiditis.

Two years afterward the patient was admitted to the Frederiksborg County Hospital. As there was then only slight paresis of all the three branches of the right facial nerve, decompression was out of the question; on the left side the palsy had subsided completely. The swelling of the upper lip had remained unchanged for the past two years. There was no regional adenitis; the salivary glands were not swollen.

Just as in the University Hospital two years before, the intraspinal pressure was increased (320 mm. of water before and 160 mm. after puncture), the cell

count was 1/3 (1 per 3 cu. mm.), the albumin content was 10 and the globulin content was 1 but, in contrast with the previous findings, Queckenstedt's sign was now distinctly positive, the pressure immediately rising from 160 to 320, and decreased function of the first and fourth cranial nerves could no longer be ascertained. Examination of the blood and roentgen study of the skull with special regard to the sella turcica showed normal conditions. The Wassermann test was negative. Lingua plicata was slight but unquestionable.

Partial excision of the upper lip was performed (figs. 1 and 2), and the microscopic examination[9] of the tissue was reported as follows:

"1. Edema: This is diffuse, medium to high grade, of even distribution, splitting the tissue elements. The standing of the edema cannot be stated with certainty; there is no fine granulation of the edematous fluid.

Fig. 2 (case 1).—Appearance of the patient after partial excision of the upper lip.

"2. Chronic inflammatory infiltration: Only slight round cell infiltration is seen, and no infiltration of the walls of venous ramifications.

"3. Vessels: It seems as if a dilatation of the lymphatic vessels can be ascertained; however, it has occurred to a slight degree only; it is more distinctly recognizable in the muscles than elsewhere in the excised tissue. The blood vessels and blood capillaries appear natural, with no thickening of the walls or thrombus formation, but here and there an accumulation of leukocytes ('leukostasis') is seen in the smaller vessels, as well as extravasation of leukocytes, which aggregate in a narrow zone immediately outside the vessel (this change resembles what is seen in hemorrhagic infarction, for example).

9. All microscopic examinations were made by Dr. Svend Petri, who also gave guidance when pathologic questions were dealt with.

"4. Nerve branches: No unquestionable pathologic alterations are seen.

"5. Muscles: The size of the fibers varies greatly between the normal ones and those showing more marked atrophy. There is an approach to an increase of the nuclei; between the fibers dense, ill defined bands of connective tissue, poor in cells, are seen to be rather widely distributed. A few small bundles of striated muscles have assumed a brighter color and apparently have lost their striation ('waxy degeneration').

"6. Squamous epithelium of the mucous membrane: The cells are not degenerated or desquamated; there is no incipient ulceration or bulla formation.

"*Microscopic Diagnosis.*—Fibrous alterations of striated muscles, only slightly characterized by chronic inflammation, with moderate to more impressive atrophy

Fig. 3 (case 2).—The patient, aged 16, shows bizarre swelling of the entire upper lip. His facial palsy had subsided. Typical lingua plicata was present. This patient was treated by partial excision of the upper lip.

of muscle fibers; edema of the mucosae of the lips, medium to high grade, with slight perivascular subacute inflammatory cellular infiltration."

CASE 2 (fig. 3).—A youth aged 16 years was admitted to the Frederiksborg County Hospital on April 7, 1945. He stated that as a child he had uncomplicated scarlatina. He mentioned no allergic predispositions and no otitis. At the age of 18 months he had right-sided peripheral facial palsy, which subsided completely in the course of one year. Two years prior to admission his upper lip began to swell greatly. This swelling was of alternating size in the beginning, but for the past six months it had remained stationary and was highly troublesome.

On admission the facial palsy had subsided completely, the entire upper lip was highly swollen, the skin was natural, there was no pulsation, the consistence

was doughy and there was no pitting on pressure. There was no swelling of regional lymphatic glands or of salivary glands. Typical lingua plicata was present. Otherwise the objective examination showed natural conditions, especially as regards the otologic and the neurologic status. Lumbar puncture revealed an increase of pressure (180 mm. before and 100 mm. after puncture). The cell count was 3/3. The albumin content was 10; the globulin content 1. The Wassermann test was negative. Roentgen study of the skull, especially as regards the sella turcica, and ophthalmoscopy revealed natural conditions.

Partial excision of the upper lip was done, and the microscopic examination was reported as follows:

"1. Edema: This is diffuse, marked, evenly distributed, splitting the tissue elements. The standing of the edema cannot be stated with certainty, because the fine granulation of the edematous fluid may be an artificial product as well as protein deposits.

"2. Chronic inflammatory infiltration: It is slight, diffuse, consisting of plasma cells and especially of leukocytes. It occurs in the corium and in the walls of a few smaller veins.

"3. Vessels: The lymphatic vessels are difficult to identify; they are hardly dilated. The fatty cells of the subcutaneous tissue that have been scattered and changed in size and shape by the edema resemble dilated lymphatic vessels. The blood vessels and the blood capillaries are normal as regards their size, content of blood and endothelium; there is no thrombus formation (compare statements in paragraph 1, however). Round cell infiltration of the walls of a few venous branches is noted.

"4. Nerve branches: No unquestionable pathologic alterations are found by means of ordinary staining methods; the sheaths of a few nerve fibers, however, seem to be somewhat thickened.

"5. Muscles: Some fibers are slightly atrophic, still without distinct increase of nuclei; between the fibers a slight and ill defined increase of connective tissue is seen here and there besides the edema.

"6. Squamous epithelium of the mucous membrane: The cells are not degenerated or desquamated; there is no incipient ulceration or bullous accumulation of fluid between the epithelium and the tunica propria.

"*Microscopic Diagnosis.*—Mucous and submucous (muscular and glandular) tissue with diffuse, medium to high grade edema and slight chronic inflammatory alterations."

On examination one year afterward the swelling of the upper lip was found to be greatly reduced, but a sufficient quantity of tissue had not been removed, and further excision will therefore be required.

CASE 3 (fig. 4).—A woman aged 30 years was admitted to the Frederiksborg County Hospital on Jan. 26, 1945. A brother had had facial palsy, which soon subsided. The patient said she had never suffered from otitis media. Fourteen years prior to admission right-sided total peripheral facial palsy suddenly developed, which was associated with gustatory disturbances but not with any acoustic or vestibular symptoms. At the same time both the upper and the lower lip began to swell, especially on the right, and this swelling remained constant from then on, without any fluctuations. The paresis subsided only partially, and a slight contracture and ticlike twitching of the right corner of the mouth developed.

Three months prior to admission massive facial palsy suddenly set in, of the left side this time, preceded by a few days' pain in and behind the left ear and associated with gustatory disturbances and buzzing in the ear but not with any acoustic or vestibular symptoms. Thereafter the patient's condition remained unchanged. Her temperature was normal.

In the left half of the face there was total peripheral facial palsy, with Bell's phenomenon; the faradic reaction was negative and the galvanic reaction positive. In the right half of the face there was partial palsy, with slight contracture and ticlike twitching of the corner of the mouth, and Bell's phenomenon was not present; the faradic and galvanic reactions were both positive.

Both lips were greatly swollen and of a firm elastic consistence; the skin did not pit on pressure; it was natural, without any pulsation. No swelling of the regional lymphatic glands or of the salivary glands was detected. Typical lingua

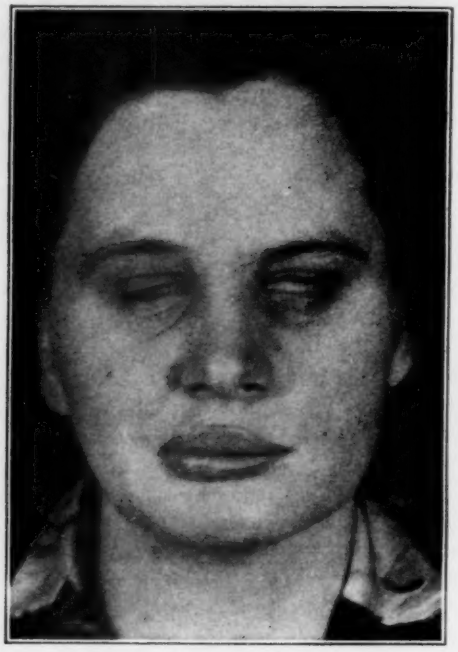

Fig. 4 (case 3).—This patient, aged 30, first presented Melkersson's syndrome fourteen years before operation. The palsy on the right side subsided partially; the swelling of the lips remained constant. Three months before admission, massive facial paresis on the left side developed, with Bell's phenomenon as shown. Typical lingua plicata was present. This patient was treated by decompression of the facial nerve and transplantation of the ilioinguinal nerve after the method of Ballance and Duel.

plicata was present. The tympanic membranes were dull but otherwise normal. Whisper was heard at more than 6 meters on each side. Neurologic examination and ophthalmoscopy revealed natural conditions. The cell count of the spinal fluid was 4/3; the albumin content was less than 10; the globulin content was less than 1. By an oversight the pressure was not measured. Roentgenograms of the skull showed blurring of the cells of the mastoid process of the left side but no unquestionable signs of destruction. The Wassermann test was negative. The blood was normal.

On Jan. 29, 1945 I performed decompression of the left facial nerve with transplantation of a section of the ilioinguinal nerve after the method of Ballance and Duel.[10]

The operation revealed highly interesting conditions. The superficial cells were large and glossy, macroscopically normal. In the depth around the antrum and in the cells between the posterior wall of the external auditory meatus and the sigmoid sinus the osseous tissue was, on the other hand, decidedly soft, and this destruction increased toward the stylomastoid foramen. The entire canal of the facial nerve from the stylomastoid foramen to the lateral semicircular canal had been destroyed, the nerve being exposed here. There was a slit in the nerve sheath, through which a piece of highly edematous nerve, 7 mm. long, was projecting, conically pointed downward, the continuity broken. A traumatic lesion was out of the question, as the microscopic examination, too, showed. This piece was resected and fixed in 10 per cent solution of formaldehyde. A nerve graft was put in, and ten months after the operation the patient began to recover mobility. This improvement is now rapidly progressing.

The microscopic examination was reported as follows: "Superficial cells: These show scantily defined subperiosteal granular bony destruction; the bony tissue is otherwise natural.

"2. Bony tissue from the area surrounding the stylomastoid foramen: Numerous osseous fragments are seen surrounded by blood and mixed with normal specific elements of the bone marrow. Small tufts of connective tissue, displaying cicatricial alterations, surround granular decayed bony tissue. The microscopic diagnosis is sequela of endosteitis with slight granular destruction of bone.

"3. Nerve and nerve sheath: The nerve sheath consists of dense collagenic connective tissue, very poor in cells, enclosing small vessels and nerve branches. In the connective tissue are slight cicatricial alterations and a few small groups of chalky deposits. The lumen of a small venous branch contains bulging, partially connected, slightly chalky connective tissue masses (possibly a thrombus in full organization). Otherwise the connective tissue is the seat of edema and hemorrhage and is covered on the inside with remnants of fibrinous exudation. There are no remnants of blood pigment, no fresh thrombus formation, in the vessels. On the whole, the nerve is of a uniform, moderately cellular structure; still it is somewhat loose everywhere. The nerve filaments stain slightly; they are of unequal size and not always of a regularly circular shape in transverse sections. The nerve filaments are split by a slight edema, and the blood capillaries are highly dilated with blood. Here and there small fresh punctiform hemorrhages are seen, small lymphocyte accumulations and a few small cicatricial strands. There is no leukocytic emigration. A special staining after the method of Mahon was made, showing degenerations of the medullary sheaths and, moreover, staining of axis-cylinders, which appear to be distinctly degenerated."

As the function of the right facial nerve was incomplete and especially as a postparalytic contracture had developed, which in Grindstein's[11] opinion is due to a reflex action on irritation of the nerve in the fallopian canal, a supposition that is rendered highly probable by the fact that in several cases I[11a] have obviated such a contracture by decompression of the facial nerve, I performed decompression of the facial nerve on the right side after the Ballance-Duel method.

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10. Ballance, C., and Duel, A. B.: Operative Treatment of Facial Palsy by Introduction of Nerve Grafts into Fallopian Canal and by Other Intratemporal Methods, Arch. Otolaryng. **15**:1 (Jan.) 1932.

11. Grindstein: Zentralbl. f. Hals-, Nasen- u. Ohrenh. **8**:299, 1926.

11a. Kettel, K.: Nord. med. **24**:1783, 1944.

The cellular system, the canal of the facial nerve and the nerve macroscopically were normal. Microscopic examination of the osseous tissue showed nothing abnormal. Immediately after the decompression the contracture subsided, the patient not becoming more paretic than before the operation, and later on mobility improved somewhat.

CASE 4 (fig. 5).—A woman aged 45 years was admitted to the Frederiksborg County Hospital on May 6, 1946. As a child she did not do well at school, owing to sleepiness. At the age of 18 she underwent a radical mastoidectomy for chronic otitis media of the left ear; from then on the ear was dry. At the ages of 18, 25 and 32 she had attacks of total right-sided peripheral facial palsy, and at the age of 38 the left side was affected, the duration being four to five

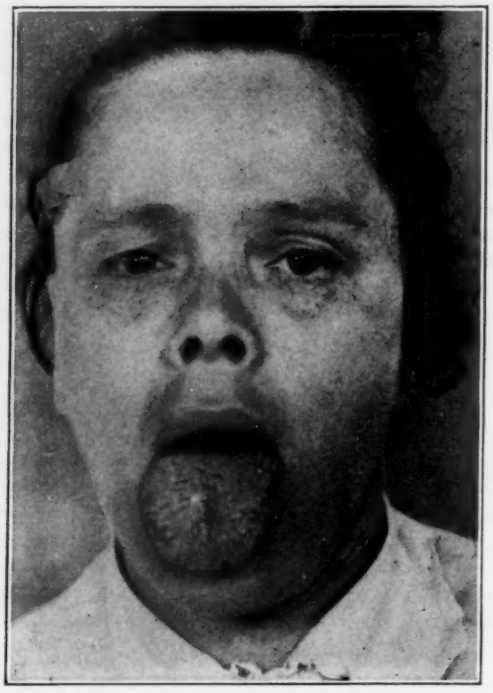

Fig. 5 (case 4).—The patient, aged 45, demonstrates lingua plicata. She presented relapsing facial palsy and pronounced pasty swelling of the entire upper lip. She was treated by decompression of the facial nerve and partial excision of the upper lip.

months each time. After occurrence of the last attack on the right side the paresis did not subside completely. After the age of 28 edema of the upper lip developed about fifteen times—only of the right side of the upper lip in the beginning, the left being involved later on. These attacks were of varying intensity, and while, to begin with, the lip was normal between attacks, the swelling in recent years had not subsided completely. The attacks were not accompanied by increase of temperature, and regional disorders or glandular swelling had never been demonstrated. No relation between the occurrence of the facial palsy and the labial edema was found.

The patient had been in the neurologic ward of the University Hospital three times with the diagnosis of chronic epidemic encephalitis of a hysterical type and Melkersson's syndrome.[12] In addition to the latter symptom complex there was an asthenic syndrome as well as hypesthesia-algesia of the entire distribution of the trigeminal nerve of the right side. Posterior pituitary injections were prescribed and were able to some extent to keep down renewed attacks of labial edema.

On admission to the hospital the patient conveyed the impression of being weak and absorbed in her disease. The function of all three branches of the facial nerve of the right side was decreased, and there were frequent tics. Bell's phenomenon was not present. Both faradic and galvanic reactions were positive. There was a pronounced pasty swelling of the entire upper lip, most marked in the right half. The color of the skin was natural, and there was no pulsation; the skin did not pit on pressure. No regional disorder, no swelling of salivary or regional glands was noted. There was marked lingua plicata. The remainder of the objective examination showed nothing abnormal apart from a dry cavity after operation on the left ear.

Owing to the relapsing facial palsy of the right side and the decreased function of the nerve, I performed decompression of the facial nerve on the right side after the Ballance-Duel method and partial excision of the upper lip on May 7. The cellular system, the canal of the facial nerve and the nerve were macroscopically normal. The postoperative course was uncomplicated. The microscopic examination of tissue excised from the lip was reported as follows:

"1. Edema: Moderate edema is seen in the zone of subcutaneous fatty tissue and in the intermuscular connective tissue.

"2. Chronic inflammatory infiltration: The corium and the tunica propria of the part of the mucous membrane that is covered with squamous epithelium display slight to moderate round cell infiltrations along the surface. The muscles, too, are the seat of scattered round cell infiltrations.

"3. Vessels: Several large cavities, covered with endothelium wholly or in part and without any contents, are seen, i. e., dilated lymphatic vessels. A couple of larger arterial branches are of normal appearance apart from minimal proliferation of the intima in one of them.

"4. Muscles: Slight muscular atrophy is seen; in some of the sections the round cell infiltration is striking.

"5. Squamous epithelium of the mucous membrane: It appears natural.

"*Microscopic Diagnosis.*—Chronic diffuse inflammation of a low to medium grade involving the skin, the mucous membrane, the subcutaneous tissue and especially the muscles; moderate edema with some dilatation of lymphatic vessels."

Case 5 (fig. 6).—A woman aged 30 years was admitted to the Frederiksborg County Hospital on May 14, 1946.

At the ages of 8 and 24 massive peripheral left-sided facial palsy of transient nature occurred without external causation, especially without exposure to cold, and was not associated with acoustic or vestibular symptoms or gustatory disturbances.

From the age of about 27 numerous (twenty to twenty-five) attacks of swelling of the left half of the upper lip occurred. They were not associated with general or regional disorder but developed at times when the patient was psychically

12. Patients 4 and 5 were referred to me by Prof. Mogens Fog, M.D., who gave me permission also to use the case books of the University Hospital.

shaken. The swelling occurred in the course of half a day, and while formerly it subsided completely in the course of a few days, later it persisted. For a year or so prior to admission she felt a swelling in the left half of the mouth, including the gums and the palate. Simultaneously with the attacks left-sided hemicrania developed. She was admitted to the neurologic ward of the University Hospital three times in the course of the period from Oct. 4, 1944 to Aug. 25, 1945. A left-sided facial contracture and myoclonia were ascertained, but no paresis; further, there was swelling of the left half of the upper lip, passing a little to the right of the median line and onto the left cheek. There were no furrows on the tongue. The remainder of the objective examination showed nothing abnormal. The cell count of the cerebrospinal fluid was 3/3; the albumin content was 6 and the globulin content 0; Pandy's test was negative. Queckenstedt's experiment showed a pressure of 100 mm. increasing to 250 mm. Roentgen study of the skull and ophthalmoscopy revealed conditions apparently natural.

The patient was extremely depressed, speaking of suicide. Preceded by great psychic unrest, a swelling of the lower lip occurred for the first time on Oct. 19, 1944, especially of the right side, and edematous fluid was seen oozing out in several parts. She was treated without any effect with injections of solutions of: a calcium salt, a salt of ephedrine, ergotamine tartrate, mersalyl and theophylline, neostigmine, methylsulfate, atropine sulfate and "insipidin" (a preparation of powdered posterior pituitary). A preparation of brewers' yeast was also given, each tablet of which supplied not less than 0.075 mg. of vitamin B_1 and 0.025 mg. of vitamin B_2. Later, boiling water was injected into the upper and the lower lip in several sittings, a treatment suggested by New and Kirch, which did not reduce the swelling but caused subsequent attacks to be less violent.

On admission to the Frederiksborg County Hospital the patient was found to have a slight facial contracture and myoclonia, but no paresis, and a firm pasty swelling of both the upper and the lower lip. The skin of the lips appeared natural; there was no pulsation and no pitting on pressure. Swelling of lymphatic or salivary glands was not present. The oral cavity, including the tongue, appeared normal. The remainder of the objective examination, including the neurologic status, showed nothing abnormal.

Because of her appearance the patient suffered from severe depression.

Partial excision of the upper and lower lips was done; in the consideration of the microscopic preparations it should be borne in mind that both lips had been given injections of boiling water; the results of the examination were reported as follows:

"Upper lip: Section of the labial mucous membrane with the adjoining submucous tissue reveals abundant striated muscular tissue but hardly any fatty cells. In the submucosa a number of small vessels and a single larger arterial branch are seen together with small, chiefly mucinous glands displaying slight round cell infiltration. The surface of the mucous membrane is folded irregularly, partially cleft, and passes on evenly to a layer of cornified squamous epithelium. The tunica propria is moderately thickened, containing a somewhat greater number of fibrils than the normal and being more densely interlaced than the normal; it also contains a greater number of capillaries. In the subpapillary stratum small numbers of round cells are seen here and there. The squamous epithelium is normal; there are no dense, hyalinized, cicatrized parts unless the hyalinization along the surface to some extent reflects cicatrization. The vessels appear natural, without alterations of the walls and without thrombi; besides the wider, blood-filled capillaries and the collapsed blood capillaries, which are empty wholly or in part, lymphatic vessels are seen here and there,

which seem to be distinctly recognizable but which cannot be termed dilated, The fibrils of the striated muscles vary slightly in size, are to some extent slightly atrophic but otherwise are not degenerated. There are no acute inflammatory changes, no remnants of hemorrhage or intermuscular proliferation of connective tissue. Only minimal edema is present.

"*Microscopic Diagnosis of Conditions in Upper Lip.*—Section of lip shows slightly indurated mucous membrane, with only slight chronic inflammatory infiltration, scattered slight muscular atrophy and comparatively scanty edema (no dilatation of lymphatic vessels).

Fig. 6 (case 5).—This woman, aged 30, had repeated swelling of the left half of the upper lip following psychic crises. She had in addition slight facial contracture and myoclonia, but no paresis and no lingua plicata. She was treated by partial excision of the upper and lower lips.

"Lower lip—(*a*) Superficial part of the excised wedge: Section of the labial mucous membrane with the adjoining submucous tissue reveals in the deeper layer a number of striated muscle fibers and a few small islands of fatty tissue. The surface of the tissue is slightly wavy; the layer of squamous epithelium is normal, without cornification. The tunica propria seems to be slightly thickened, more dense, containing more fibrils than the normal and also a greater number of vessels. Here and there lymphatic vessels are seen, some of which are highly dilated; this applies to one of the vessels in particular, which fills

out one of the connective tissue papillae completely and projects into the layer of squamous epithelium. No ulceration, no coating is seen. In the deeper parts of the tunica propria a few dilated lymphatic vessels are seen. The tunica propria displays a diffuse round cell infiltration of a median—sometimes higher-grade. In several of the muscle bundles a slight decrease of some of the fibrils is seen.

"(*b*) Deeper part of the excised wedge: This piece of tissue represents a section of striated muscles. Scattered small bundles of fibrils are moderately atrophic. Intermuscularly, and especially at one edge of the preparation, presumably in the submucous layer, there is slight to more marked edema but no dilated lymphatic vessels. There is diffuse slight chronic inflammatory reaction.

"*Microscopic Diagnosis of Conditions in Lower Lip.*—Section of lip shows very slightly indurated mucous membrane with a median grade of chronic inflammatory infiltration, diffuse slight muscular atrophy and moderate edema, chiefly in the submucosa (scattered; in parts, great dilatation of lymphatic vessels)."

In spite of the preceding injection of boiling water and despite the excision the patient still reported a slight acute exacerbation ten days after the operation, but the swelling subsided again in the course of a few days.

CLINICAL FEATURES

Most frequently the disease begins in childhood or youth with peripheral facial palsy; 3 of my patients were women, 2 were men, and in all of them the disease had its onset with facial palsy before the eighteenth year (at the ages of 2, 1½, 16, 18 and 8 years, respectively). Four of the 5 patients of Ekbom and Wahlström were men, and in all these patients, too, the disease had begun with palsy before the eighteenth year.

The swelling of the face may occur simultaneously with the facial palsy, but in many cases a long time, up to many years, will pass before this symptom appears. In only 1 of my patients did the swelling of the face occur at the same time as the facial palsy; in the remaining 4 patients the paresis appeared from ten to nineteen years before the face became swollen. Of the patients observed by Ekbom and Wahlström, 2 had swollen lips soon after the occurrence of the paresis; in 3 the swelling did not appear until from two to seven years later.

The swelling sets in without any preceding general disorder, and local or regional disorders especially cannot be demonstrated. It should be noted that some of the patients observed that the swelling began after they had been exposed to cold. It is localized to the eyelids, the nose, the chin and especially to the lips, which may become three to four times thicker than the normal. In 3 of my patients the swelling was localized to the upper lip, in 2 of them (cases 3 and 5) to both the upper and the lower lip, which was also observed in 4 of the patients of Ekbom and Wahlström. Whereas an isolated swelling of the lower lip has not been observed by me, Ekbom and Wahlström have noted it in 1 case. In 1 of my patients (case 5) the swelling covered part of the chin. At first the swelling may be transient, later passing on to the

chronic stage, or it may be "primary chronic" (Ekbom and Wahlström). In 2 of my 5 patients the swelling was "primary chronic," this being the case also in 4 of the patients of Ekbom and Wahlström, with a tendency, however, toward variation and progression. The skin appears natural; it does not pit on pressure and there is no pulsation and no demonstrable regional disorder or adenitis.

The swelling of the face has been compared to Quincke's edema, and indeed there are many points of resemblance, especially as far as the group is concerned in which the swelling is transient to begin with; it is, however, striking that in Melkersson's syndrome the swelling appears only in the face and never elsewhere.

As to the anomaly of the tongue, 1 of the patients of Ekbom and Wahlström had had lingua plicata since childhood, a long time before the other symptoms appeared, and in another case the same symptom was found in the patient's mother and sister. The authors have therefore expressed the belief that in Melkersson's syndrome one has to do with genuine lingua plicata and not with swelling of the tongue coordinate with that of the lip, and they have emphasized that there is probably a hereditary predisposition. It is considered to be a harmless anomaly, but it has also been regarded as a "stigma degenerationis." The furrowed tongue, however, may swell simultaneously with the lips, this having occurred in 2 of the patients observed by Ekbom and Wahlström. Four of my patients had typical lingua plicata, but the tongue did not swell in any of the 5 patients; 2 of the patients of Ekbom and Wahlström had this symptom to a marked degree, while in 3 the tongue was moderately, though distinctly, furrowed. It is still an open question why facial palsy and facial edema develop in patients with lingua plicata.

The facial palsy is always of a peripheral type; it may be bilateral, and was in patients 1 and 3. It may relapse; this was seen in patient 1 three times, in patient 4 three times and in patient 5 twice. It may appear together with the swelling of the lips but, as has been mentioned, it may also precede this symptom by many years; precedence was observed in 4 of my patients. It cannot be distinguished clinically from Bell's palsy, for, like the latter, it may occur suddenly or develop gradually, and be complete or partial. In all my 5 patients the onset was sudden and massive. Several of the patients of Ekbom and Wahlström, as well as my first patient, are said to have been exposed to the influence of cold immediately before the onset of the paresis. Such exposure is frequently noted in connection with Bell's palsy but the importance of it is the subject of much difference of opinion.

PATHOLOGIC FEATURES

Pathologic Anatomy.—For practical reasons the facial palsy will be mentioned first. The paresis in Melkersson's syndrome cannot be clinically distinguished from Bell's palsy, which, however, does not

make the matter more lucid, because Bell's palsy is no etiologic entity but, in fact, comprises all cases of peripheral facial palsy in which it has been impossible to demonstrate any cause—first and foremost, nonsuppurative otitis and syphilis.

The question of the pathology of Bell's palsy will be thoroughly dealt with in this periodical in the near future, for which reason only a few details will be mentioned now.

Decompression of the facial nerve after the method of Ballance and Duel has been performed on 50 patients, and in 11 of these a decay of the osseous system was found in the form of widespread destruction, especially around the stylomastoid foramen, whereas in the remaining patients the air cell system was macroscopically normal. The facial canal proper was completely soft and necrosed in 18 of 50 patients and hard like ivory in the others. Lastly, with regard to the nerve, it was found to be distinctly edematous in 28 of 50 patients, all minor alterations not being considered.

Patient 3 in the present communication represents the extremes; for, considering her right facial nerve, one would say that she was of the group in which the operation did not reveal anything abnormal, whereas on the left side a thorough osseous necrosis with considerable alterations of nerve and nerve sheath could be demonstrated. In patient 4, on the other hand, nothing macroscopically abnormal was found in the cells of the mastoid process in the canal of the facial nerve or in the nerve.

Little is known from the point of view of pathologic anatomy about the swelling of the face. New and Kirch wrote about this question:

> Specimens of tissue were taken for biopsy in a few cases before treatment, but nothing was found other than edematous tissue containing lymphocytes.

Ekbom and Wahlström made a microscopic examination in 1 case from which they drew certain conclusions:

> The preparation consists of striated muscles and connective tissue with round cell accumulations. The muscular bundles are not, as in normal muscles, placed side by side but are separated by cavities. The latter are found to be empty, when the sections are examined, and they may possibly be dilated lymphatic spaces. Further, under the epithelium a sclerotic connective tissue can be observed containing numerous ectatic lymphatic vessels. Diagnosis: muscle fibers and connective tissue with dilated lymphatic vessels.

Judging by this preparation, the authors continue, the swelling, in the chronic stage, should be determined by dilated lymphatic vessels and sclerotic connective tissue. Next they discuss the following two possibilities: In the acute stage there is either edema, after which, following repeated relapses, chronic inflammatory changes of, and irreversible injury to, the lymphatic vessels occur, with resulting lymphatic stasis, or the latter is present right from the onset and particularly so in the "primary chronic" condition. They adopt the latter view.

My examinations, however, gave a different result. The operations were performed with the patient under evipal anesthesia.

What is found (see the table) is a more or less pronounced edema, especially of the subcutis and the muscles, chronic inflammatory infiltrations, slight dilatation of the blood vessels, slight muscular atrophy and, in a few cases, a partially marked proliferation of the intermuscular connective tissue. But the lymphatic vessels are either not dilated at all (and it should be noted that this applies to the 2 cases in which the swelling of the lip was most marked) or they are only slightly dilated; only in 1 of 6 preparations were a few highly dilated lymphatic vessels found.

In the 2 cases in which the swelling of the upper lip was most marked (1 [fig. 1] and 2 [fig. 3]) the microscopic examination showed beyond dispute that the swelling was due to marked edema, whereas the lymphatic vessels of the lip were normal. In 2 cases (4 [fig. 5] and 5 [fig. 6]) the macroscopic swelling of the upper lip was smaller and correspondingly the edema was not so impressive in the microscopic examination; on the other hand, the round cell infiltration was more prominent, and only in 1 of these cases were the lymphatic vessels somewhat dilated—in the other case they were completely normal.

This means that in 3 of 4 cases the macroscopic swelling of the upper lip was due to edema and to chronic inflammatory reactions but not to lymphatic stasis; a few dilated lymphatic vessels could be demonstrated only in case 4.

The preparations made from excised tissue of the lower lip were from patient 5, who suffered from fairly frequent exacerbations of her chronic labial swelling. A wedge comprising the red of the lip was removed, passing downward deep into the muscles, the base of the wedge turning upward. On microscopic examination of the base the squamous epithelial coating was found to be normal and without any cornification. The tunica propria seemed to be slightly thickened and to contain more fibrils than it would normally; it also seemed to contain rather a greater number of vessels. Here and there lymphatic vessels were seen, some of them being greatly dilated, especially a single vessel which completely filled out one of the larger connective tissue papillae and projected into the squamous epithelium. In the apex of the wedge, on the other hand, the lymphatic vessels were found to be normal.

Thus in this patient's lower lip there was no universal dilatation of lymphatic vessels, only some of the latter being greatly dilated.

It is an interesting fact that when this patient first had a swelling of her lower lip an "edematous fluid" could be seen oozing out in several parts (eighteen months before the excision), and it is not improbable that a rupture of ectatic lymphatic vessels similar to that observed in the microscopic examination had taken place then.

Summary of Microscopic Data on Five Cases of Melkersson's Syndrome in Which the Treatment was Partial Excision of the Lips

Patient	Figure	Age, Yr.	Time in Years from Beginning of Enlargement of the Lip to Operation	Macroscopic Enlargement of the Lip	Edema *	Chronic Inflammatory Infiltration	Blood Vessels		Lymphatic Vessels *		Muscles *		Increase of Intermuscular Connective Tissue	Epithelium	
							Normal	Dilated	Normal	Dilated	Normal	Atrophic		Normal	Pathologic
1	1-2	18	2	+++	++, +++	(+)	+	.....	.....	(+)	.....	—, ++	+++	+	.....
2	3	16	2	+++	++, +++	(+)	+	....?.	+	.....	.....	(+)	+	+	.....
4	5	45	17	+	+	(+), +	+	.....	.....	—, ++	.....	(+)	—	+	.....
5	6	30													
Upper lip.....			3	+	(+)	(+)	.....	+	+	.....	.....	(+)	—	+	.....
Lower lip, superficial part of wedge			1½	++	—	++, +++	.....	+	.....	—, +++	.....	(+)	—	+	.....
Lower lip, deeper part of wedge			1½	++	+, ++	++	+	.....	+	.....	.....	+	—	+	.....

* Two signs in a column indicate that the reaction in different parts of the slides varied between these extremes. Key: (—) no such condition present; (+) slight degree; +, moderate degree; ++, medium degree; +++, high degree. A combination such as —, ++ or ++, +++ describes conditions seen in different parts of the field.

This highly dilated lymphatic vessel was localized to a part immediately below the epithelium, and it should be noted that the unquestionably dilated lymphatic vessels in the cases reported by Ekbom and Wahlström had the same localization. With respect to their suggestion that the cavities between the muscular bundles in their preparation might be dilated lymphatic vessels, it must be observed that an endothelial coating of the latter had not been demonstrated, and, further, it should be borne in mind that the lipoid contents of the fat cells are dissolved when the section is prepared, leaving small round cavities and that such cavities may have been mistaken for lymphatic vessels.

This much seems to be certain if I may judge from my preparations: In the chronic stage there is no chronic lymphatic stasis as supposed by Ekbom and Wahlström, but chronic edema. No microscopic examinations have been made during the acute stage, but the clinical observation just referred to seems to indicate that simultaneously with the edema there is dilatation of at least a few lymphatic vessels.

Nothing at all is known at present from the standpoint of pathologic anatomy about the anomaly of the tongue.

Pathogenesis.—Now, what is the pathogenesis? Much seems to indicate that the primary and central feature of the pathogenesis of Bell's palsy, from which the facial palsy occurring in Melkersson's syndrome cannot be clinically distinguished, probably is a disturbance of circulation, a disorder of the vasa nervorum (Pollak [13]; Audibert, Mattei and Paganelli [14]); the nerve is paralyzed because there is a block of the blood vessels, and as a consequence of this it secondarily becomes edeatous (*Quellung*) and is compressed in the facial canal. It has already been mentioned that such edema of the facial nerve can frequently be demonstrated when one operates on patients suffering from Bell's palsy (Ballance and Duel; Collier [15]; Morris [16]; Cawthorne [17]; Kettel [11a]). Both the centrifugal and centripetal impulses are now blocked, which, in addition to the paresis, gives rise to inactivity and resultant atrophy of the facial nuclei, followed by descending degeneration of the peripheral neuron, if the vascular supply of the nerve is blocked for a long enough time. The destructive processes which I found in the mastoid process in 11 of 50 patients, as described earlier in this article, and in the wall of the facial canal are a collateral reaction to the same noxa (ischemic bony necrosis).

In my opinion the swelling of the face must also be regarded as angioneurotic edema. In Melkersson's syndrome the consistence of

13. Pollak, E., in Alexander, G., and Marburg, O.: Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1924, vol. 2, pt. 1, p. 1.

14. Audibert, V.; Mattei, C., and Paganelli, A.: Presse méd. **1**:1049, 1936.

15. Collier, J.: Lancet **2**:91, 1940.

16. Morris, W. M.: Lancet **1**:429, 1938.

17. Cawthorne, T.: Laryngoscope **56**:653 (Nov.) 1946.

the lips has quite the character of angioneurotic edema, and the vigorous development of intermuscular connective tissue which was seen in the patient with the most marked swelling in my series is also characteristic of such edema. The resemblance to Quincke's angioneurotic edema has been referred to repeatedly in the literature, but in every instance it has been stated that "Melkersson's swelling" was confined to the face in contrast with Quincke's edema.

There is, however, a fact which has not been referred to previously: The patients suffer from paralysis of the voluntary muscles corresponding to the part innervated by a particular nerve, namely, the facial nerve, and in addition they suffer from edema which is confined to the distribution of the same nerve. Therefore it may be presumed that the vasomotor fibers of the face follow a shorter or longer course in or along the facial nerve. Paralysis of the sympathetic fibers involves both paresis of the vascular supply of the facial nerve, resulting in facial palsy, and paresis of the vessels of the face, followed by edema.

Nothing is known about the pathogenesis of the anomaly of the tongue. It is stated that the tongue may swell simultaneously with the lips, but this symptom may also be present a long time before the swelling of the face begins, and the anomaly of the tongue may also be hereditary. In the case in which swelling of the tongue is seen simultaneously with that of the lips, it is natural to look for a common pathogenesis. If it be accepted that vasomotor fibers take a course in or along the facial nerve and that paralysis of these fibers brings about the swelling of the face, then it must be borne in mind that there are several anastomoses between the facial nerve and the glossopharyngeal nerve, so that the vasomotor fibers to the tongue may fail at the same time.

ETIOLOGIC CONSIDERATIONS

Ekbom and Wahlström, who considered the disorder as chronic lymphatic stasis, examined the various etiologic possibilities (allergy, syphilis, venereal lymphogranuloma, lymphogranulomatosis benigna, basal diseases of the throat and the teeth) but arrive at the result that the disease is completely puzzling.

Buch suggested the possibility that in his case the cause was basal arachnoiditis.

Since in my opinion the facial palsy, as well as the swelling of the face, is an angioneurotic phenomenon, the etiologic factor must consequently be sought among such factors as may cause a disturbance of the regulation of the vasomotor nerves.

DIFFERENTIAL DIAGNOSIS

The syndrome is characteristic; in the acute stage the disease has been mistaken for a parulis and for erysipelas; in the chronic stage it has been mistaken for acromegaly.

THERAPY

New and Kirch recommended that boiling water be injected into the swollen tissue and that radium treatment be used, followed by excision of that part of the subcutaneous tissue. They stated that it is important to carry out the preliminary treatment before the excision in order to counteract the tendency toward renewed swelling of the lip. If this is not done, one runs the risk that the patient's condition may become worse after the operation than it was before.

In case 1, however, only excision was done, and the result was satisfactory; in case 5, despite preceding injection of boiling water and subsequent partial excision, there occurred another attack of acute edema, which, however, subsided.

The significance of this is, partly, that excision may be done without preceding injection of boiling water or radium treatment without running the risk of renewed swelling of the lip and, partly, that the injection referred to by no means protects the patient against relapse.

As to Melkersson's facial palsy, I shall follow the same lines of treatment as in Bell's palsy, owing to the belief that it is identical with the latter; I have taken this attitude on the basis of 50 decompression operations. In fresh cases the patient should be observed for about two months; if there are no signs of beginning mobility after that period, decompression of the facial nerve should be performed in accordance with the Ballance-Duel method. Moreover, the same treatment is indicated if the spontaneous mobility has ceased before complete restitution has been obtained, and in case of relapsing palsy.

SUMMARY

Melkersson's syndrome is a characteristic facies morbi comprising the following triad: chronic swelling of the face, peripheral facial palsy, which may be bilateral and which may tend to relapse, and lingua plicata. As a rule, the disease begins in childhood or youth with an attack of peripheral facial palsy, which may be bilateral and possibly may relapse; either simultaneously or later (sometimes not until several years later) a swelling of the face occurs, especially localized to the lips. The tongue is distinctly furrowed (lingua plicata).

Both clinically and pathologicoanatomically the facial palsy shows close correspondence to Bell's palsy. Two of the patients with bilateral palsy were operated on after the method of Ballance and Duel (decompression of the facial nerve), and while in the first patient nothing macroscopically abnormal could be demonstrated in the bone or the nerve on one side, extensive changes both of the mastoid process and the facial nerve were found on the other side. In the other patient there were no abnormal macroscopic findings in the cells of the mastoid process, the canal of the facial nerve or the nerve.

Much seems to indicate that the palsy is due to a disturbance of the regulation of the vasa nervorum of the facial nerve.

During the chronic stage the swelling of the face is due to chronic edema, which has all the characteristic features of angioneurotic edema.

The facial palsy and the swelling of the face occur within the parts innervated by a particular nerve, the facial nerve, for which reason it is supposed that in these patients this nerve, besides supplying the voluntary facial muscles with motor fibers, also conducts vasomotor fibers. Decreased function of the former results in facial palsy, whereas disturbance of the innervation of the latter causes neurotrophic facial edema.

The treatment consists in excision of superfluous tissue, preceded, if required, by an injection of boiling water into the swollen tissue, plus radium treatment (New and Kirch).

If after two months' observation the facial palsy has not displayed signs of beginning spontaneous regeneration, or if the spontaneous regeneration has come to a standstill before complete restitution has been obtained, or if the palsy relapses, decompression of the facial nerve according to the Ballance-Duel method must be done.

Frederiksborg County Hospital.

RELATION OF VARIOUS FUNGI TO OTOMYCOSIS

FREDERICK T. WOLF, Ph.D.
NASHVILLE, TENN.

IT HAS BEEN over a century since Mayer (1844) originally described a disease of the external canal of the ear caused by a fungus. At the present time the literature concerning this type of infection, known as otomycosis, contains reports of numerous cases in which a great variety of fungi were isolated. Reports of such cases describe symptoms and signs which were exceedingly diverse and suggest methods of treatment as numerous as the fungi isolated.

It is believed that a factor of great importance in explaining this chaotic state of the medical knowledge with respect to otomycosis has been the failure to recognize that the disease is not an entity but, rather, may embrace a number of different diseases caused by widely diverse groups of organisms. In this respect, fungi are seldom isolated from the ear in pure culture but are frequently accompanied by bacteria. It is not definitely known, therefore, whether the fungi are primary or secondary invaders. Moreover, in many cases external otitis appears to be of purely bacterial origin, the evidence indicating that Pseudomonas aeruginosa (Bacillus pyocyaneus), Staphylococcus, Streptococcus and diphtheriods may be of etiologic significance.

Furthermore, the fungi commonly isolated from the infected external canals are, with few exceptions, not types whose pathogenicity is definitely known. Rather, they are, for the most part, common organisms of the soil which are able to lead a saprophytic existence outside the body and can be easily isolated from dust or air. Thus, caution should be exercised in ascribing pathogenesis to them.

Few attempts have been made to establish the pathogenicity of the fungi recovered from diseased ears by animal inoculation. The studies of Carnevale-Ricci (1927), Africa (1933), Enlows (1935) and others, while representing a step in the proper direction, leave some doubt as to the ability of the organisms concerned (Aspergillus fumigatus and Lichtheimia corymbifera) to invade healthy epithelium and produce symptoms entirely comparable to those observed in man. Further, many additional species of fungi remain uninvestigated in this respect. Also, there have been no investigative studies to determine whether these fungi may produce lesions not by directly invading tissue but by

discharging toxic by-products as they maintain a possibly saprophytic growth in the cerumen and seborrheic material of the ear.

Before the studies indicated in the foregoing paragraph can be successfully achieved, it will be necessary to identify accurately every fungus isolated in a case of otomycosis. That this will be difficult is readily recognized, since the literature is full of instances in which the fungi reported were either partially identified or misidentified. In only a handful of reports are statements as to the identity of the fungi supported by the opinions of competent mycologists or accompanied by sufficient descriptions, drawings or photographs to make their identity apparent. Many often-quoted publications which name various species of fungi as of etiologic significance fail entirely to refer to authoritative articles to which an investigator may turn to find support for the quoted statement.

Since the literature concerning otomycosis is widely scattered, with few attempts at summarization, not many articles relating to the subject have been availiable to individual workers. In order to provide a basis for the possible determination of the relation of various fungi to otomycosis, the literature has been sifted and a bibliography of the important contributions compiled.

The organisms reported in the literature as causative belong to the Aspergillaceae, Mucoraceae, Actinomycetaceae, dermatophytes, yeastlike fungi and a great variety of miscellaneous fungi imperfecti. The Aspergilli, reputedly accounting for about 90 per cent of the reported cases, have been grouped according to the classification of Thom and Raper (1945). As no modern monographic treatments of many of the other groups exist, these fungi have been listed only, without attempting to describe the individual groups.

The list of fungi reported to cause otomycosis is as follows:

ASPERGILLACEAE

ASPERGILLUS

A. ASPERGILLUS FLAVUS GROUP. Conidia yellow-green, conidiophores rough walled. Sterigmas variable, in one or two series, conidia roughened.

1. *Aspergillus flavus* Link: Cassells (1874), Siebenmann (1889), Ferreri (1894), Hatch and Row (1900), Lewis (1927), Gordon (1928), Drennan (1928), Trimarchi (1929), Dunlap (1937), Mood (1938), Pittenger (1938), Terasi (1939), Minchew, Collins and Harris (1940), Fujishiro (1941), Keeney and Broyles (1943), Iriarte (1943), Simon (1945), Salvin and Lewis (1946).

2. *Aspergillus oryzae* (Ahlburg) Cohn.: ?Talice and Mackinnon (1932), Mood (1938).

B. ASPERGILLUS GLAUCUS GROUP. Perithecia present, yellow, with eight-spored asci, ascospores with an equatorial furrow. Conidial heads radiate to columnar, green, with sterigmas in one series. Conidia elliptic to subglobose, roughened.

3. ***Aspergillus montevidensis*** Talice and Mackinnon: Talice and Mackinnon (1931, 1932).

4. ***Aspergillus repens*** (Corda) de Bary: Siebenmann (1889), Marena (1929),

5. ?***Aspergillus glaucus*** Link: In a number of cases of otomycosis, Aspergilli with the characteristics of the A. glaucus group have been reported under the name A. glaucus. A. glaucus is not a valid species according to Thom and Raper (1945), and the descriptions given are not sufficiently detailed to indicate identity of the species, although these forms can be assigned to the A. glaucus group with certainty. The following reports have been maintained under the name A. glaucus as a matter of convenience: Hassenstein (1869), Knapp (1869, 1871), Moos (1872), C. H. Burnett (1877, 1889), Hatch and Row (1900), Bar (1904), Forsyth (1924), Penido Monteiro (1925), Trimarchi (1929), McBurney and Searcy (1936), Mood (1938), Senturia and Wolf (1945), Simon (1945).

C. ASPERGILLUS FUMIGATUS GROUP. Conidial heads columnar, green. Vesicles flask shaped, the upper half fertile. Sterigmas in one series; conidia globose, echinulate, green.

6. ***Aspergillus fischeri*** Wehmer: Lindt (1889).

7. ***Aspergillus fumigatus*** Fresenius: Hallier (1870), Siebenmann (1889), Hatch and Row (1900), Liébault (1922), Carnevale-Ricci (1927), Citelli (1927), Fort (1927, 1928), Ciampi (1928), Thévenard (1928), Castellani (1928), Trimarchi (1929), Peri (1929), Marena (1929), Cavadas (1930), Castellani (1932), Enlows (1935), McBurney and Searcy (1936), Dunlap (1937), Terasi (1939), Minchew, Collins and Harris (1940), Fujishiro (1941), Dobes (1943), Iriarte (1943), Keeney and Broyles (1943), Senturia and Wolf (1945), Simon (1945), Magalhaes (1945).

D. ASPERGILLUS NIDULANS GROUP. Conidial heads columnar, dark green. Sterigmas in two series. Conidia echinulate. Perithecia present, the ascospores red, with equatorial bands. Assignable to this group are a number of cases due to an organism designated as A. flavescens, which is not identifiable with any species accepted at the present time.

8. ?***Aspergillus flavescens*** Wreden: Wreden (1867), Roosa (1870), Blake (1871), Liljenroth (1872), Bezold (1873), Bowen (1874), C. H. Burnett (1874), Wreden (1874), Hall (1877), C. H. Burnett (1878, 1879, 1880), Galloway (1929).

9. ***Aspergillus nidulans*** (Eidam) Wint: Wreden (1874), Siebenmann (1889), Marena (1929), Trimarchi (1929), Enlows (1935), Mood (1938), Terasi (1939).

E. ASPERGILLUS VERSICOLOR GROUP. Conidial heads hemispheric to globose, radiate, green or blue-green, with sterigmas on the upper one half or three fourths of the head, in two series. Conidia echinulate.

10. ***Aspergillus sydowi*** (Bainier and Sartory) Thom and Church: ?Hallier (1869), Terasi (1939), Minchew, Collins and Harris (1940), Senturia and Wolf (1945).

11. ***Aspergillus versicolor*** (Vuillemin) Tiraboschi: S. M. Burnett (1881), Terasi (1939).

F. ASPERGILLUS TERREUS GROUP. Conidial heads columnar, cinnamon, bluff or light flesh in color. Vesicle hemispheric, the upper one half to two thirds covered by sterigmas. Sterigmas in two series. Conidia small, smooth, globose to slightly elliptic.

12. *Aspergillus terreus* Thom: Langeron (1922), Chisolm and Sutton (1925), Terasi (1939), Fujishiro (1941), Simon (1945), Salvin and Lewis (1946).

G. ASPERGILLUS CANDIDUS GROUP. Conidial heads white, globose to columnar. Sterigmas in two series. Conidia globose, smooth.

13. *Aspergillus candidus* Link: ?Siebenmann (1889), Hatch and Row (1900), Pound (1901), Cook (1903), Penido Monteiro (1925), Ch'in (1935).

H. ASPERGILLUS OCHRACEUS GROUP. Heads sulfur yellow to ocher, globose or radiate. Sterigmas in two series.

14. *Aspergillus ochraceus* Wilhelm: Bellucci (1925), Terasi (1939).

I. ASPERGILLUS NIGER GROUP. Conidial heads black, globose Vesicles globose. Sterigmas in two series. Conidia rough.

15. *Aspergillus niger* van Tieghem: Wreden (1867), Bezold (1870), Roosa (1870), Steudener (1870), Blake (1871), Bezold (1873), Kilpatrick (1873), Bizzell (1874), Cassells (1874, 1875), Wreden (1874), Hall (1877), Strawbridge (1878), C. H. Burnett (1878, 1879), Torrance (1880), Rush (1881), Theobald (1881), C. H. Burnett (1889), Siebenmann (1889), Ferreri (1894), Theobald (1898), Hatch and Row (1900), Guarnaccia (1904), Bar (1904), Stout (1912), Bellows (1916), Koenig (1920), Forsyth (1924), Anderson (1925), Motta (1925), Lewis (1927), Citelli (1927), Miller (1927), Weill and Rubinstein (1927), Fort (1927, 1928), Gordon (1928), Gable (1929), Galloway (1929), Marena (1929), Peri (1929), Salzberger (1929), Trimarchi (1929), Searcy and McBurney (1929), Cavadas (1930), Motta (1931), Talice and Mackinnon (1932), Amstutz (1934), Kulvin (1935), Enlows (1935), Gomez, de Cisneros and Vallejo Vallejo (1935), McBurney and Searcy (1936), G. E. Martin (1937), Tempéa (1937), Dunlap (1937), Simms (1937), Chiniara (1937), Pittenger (1938), Engel (1938), Mood (1938), Reboucas (1939), Terasi (1939), Minchew, Collins and Harris (1940), Felderman (1940), Dart (1940), Mitra (1940), Cavalcanti (1941), Fujishiro (1941), Iriarte (1943), Keeney and Broyles (1943), Simon (1945), Senturia and Wolf (1945), Salvin and Lewis (1946).

16. *Aspergillus phoenicis* (Corda) Thom.: Cramer (1859).

17. *Aspergillus macfiei* Dodge: Macfie (1921).

J. ASPERGILLUS WENTII GROUP. Conidial heads large, globose, yellow-brown, orange or umber. Sterigmas in two series. Conidia elliptic, smooth or roughened.

18. *Aspergillus wentii* Wehmer: Trimarchi (1929), McBurney and Searcy (1936).

K. ASPERGILLUS SP. INDET. The following reports concern cases in which otomycosis appears to be due to Aspergilli that are not identifiable as to species:

Mayer (1844), Pacini (1851), Schwartze (1867, 1868), Hallier (1869), Böke (1869), Green (1868, 1869, 1870), Nölting (1870), Bezold (1873), Bürkner (1883), Hatch and Row (1900), Maurin (1903), Cheatle and Emery (1919-1920), Cheatle (1920), Forsyth (1924), Motta (1925), W. D. Gill (1935), Karp (1935), Pittenger (1938), Minchew, Collins and Harris (1940), Dart (1940), Yates (1944), Basil-Jones (1945), Nelson (1945), Syverton, Hess and Krafchuk (1946).

PENICILLIUM

19. *Penicillium bicolor* Fries: Motta (1925).

20. *Penicillium crustaceum* Fries: Pound (1901), Motta (1925), Marena (1929).

21. *Penicillium eborinum* Sasaki: Sasaki (1939), Fujishiro (1941).

22. *Penicillium jantho-citrinum* Biourge: Sasaki (1939), Fujishiro (1941).

Penicillium sp. indet.: Siebenmann (1889), Hatch and Row (1900), Chisolm and Sutton (1925), Motta (1925), W. D. Gill (1935), Mood (1938), Minchew, Collins and Harris (1939, 1940), Dart (1940).

SCOPULARIOPSIS

23. *Scopulariopsis brevicaulis* (Sacc.) Bainier: Pinkerton and MacQuiddy (1936).

24. *Scopulariopsis sasakianus* Sasaki: Sasaki (1939).

MUCORACEAE

LICHTHEIMIA

25. *Lichtheimia corymbifera* (Cohn) Vuillemin: Hückel (1886), Siebenmann (1889), Graham (1890), Motta (1925), Carnevale-Ricci (1927), Marena (1929).

26. *Lichtheimia ramosa* Vuillemin: ?Jakowski (1889), Castellani and Chalmers (1919), Castellani (1928, 1932).

Lichtheimia sp. indet.: Iriarte (1943).

MUCOR

27. *Mucor circinelloides* van Tieghem: Dowding and Levey (1939).

28. *Mucor mucedo* L.: Böke (1869), Bar (1904), Galloway (1929).

29. *Mucor pusillus* Lindt: ?Jakowski (1889).

30. *Mucor racemosus* Fresenius: Pound (1901).

Mucor. sp. indet.: Wreden (1874), Siebenmann (1889), Bar (1904), Chisolm and Sutton (1925), Wilson (1928), Basil-Jones (1945).

RHIZOPUS

31. *Rhizopus nigricans* Ehrenberg: Pound (1901).

Rhizopus sp. indet.: Dart (1940).

SYNCEPHALASTRUM

32. *Syncephalastrum racemosum* Cohn: I have observed a case of otomycosis due to this organism, hitherto unknown in this connection.

YEASTLIKE FUNGI

33. *Candida albicans* (Robin) Berkhout: ?Bar (1904), Simms (1939), Dobes (1943), Simon (1945).

34. *Candida tropicalis* (Castellani) Berkhout: Syverton, Hess and Krafchuk (1946).

35. *Monilia rhoi* Castellani: Castellani and Chalmers (1919), Castellani (1928, 1932).

36. *Mycotoruloides alba* Sasaki: Sasaki (1939).

37. *Mycotoruloides alba* var. *furcellata* Sasaki: Sasaki (1939).

Candida (Monilia), sp. indet.: Dart (1940), Daggett (1942), Basil-Jones 1945). Saccharomyces sp. indet.: Forsyth (1924), ?Basil-Jones (1945).

DERMATOPHYTES

Achorion sp. indet.: ?Whalen (1938).

38. *Trichophyton violaceum* Sabouraud: Basil-Jones (1945).
Trichophyton sp. indet.: ?Bar (1900), Dart (1940), Davis (1943).

ACTINOMYCETACEAE

39. *Actinomyces israeli* Dodge: Salvin and Lewis (1946).
Actinomyces sp. indet.: Riviere and Thevenot (1904), Döderlein (1925), Risch (1939), Syverton, Hess and Krafchuk (1946).

40. *Streptomyces albus* (Rossi-Doria) Waksman and Henrici: Odom, Rodenberg and Schain (1944). I have observed a single case of severe otitis externa from which this organism was isolated.
Sporothrix sp. indet.: Bahre and Hansen (1929).
Streptothrix sp. indet.: Cheatle (1920), R. C. Martin (1931).

MISCELLANEOUS FUNGI

41. *Alternaria tenuis* Nees: Motta (1925).
Alternaria sp. indet.: Dart (1940).

42. *Alysidium rufescens* Fresenius: Pound (1901).

43. *Bargellinia monospora* Borzi: Pound (1901).

44. *Coremium bicolor* (Web.) Pound and Clements: Pound (1901).

45. *Graphium penicilloides* Corda: Hassenstein (1869), Siebenmann (1889).

46. *Hormodendrum pyogenes* Machado and Romeo Filho: Machado and Romeo Filho (1940).

47. *Malassezia furfur* (Robin) Baillon: Yates (1944).

48. *Mollisia auriculae* (Garovoglio) Saccardo: Pound (1901).
Monosporium sp. indet.: Belding and Umanzio (1935).
Monotospora sp. indet.: Salvin and Lewis (1946).

49. *Stemphylium polymorphum* Bonorden: Pound (1901).

50. *Tilletia levis* Kühn: Castellani and Chalmers (1919).

51. *Trichothecium roseum* Link: Steudener (1870), ?Bar (1904).

52. *Ustilago carbo* Tulasne: Castellani and Chalmers (1919).

53. *Verticillium graphii* Bezold: Siebenmann (1889), Herzog (1895).

SUMMARY AND CONCLUSIONS

At least 53 different species of fungi have been reported to cause otomycosis. Because of the dubious accuracy of the identification of many of these organisms and the lack of conclusive evidence of their pathogenicity, the literature does not provide a valid evaluation of the problem of otomycosis. It is further apparent that if future studies continue in the paths of the older ones, the problem of the etiologic factors of otomycosis will remain forever obscure.

In order to bring present day understanding of otomycosis to the level attained with other mycotic diseases, future studies must include:

1. Determinations of the normal microbiologic flora of the aural canals of a large number of healthy human subjects.

2. Competent microbiologic identifications of cultures obtained from diseased ears. This is essential if any value is to be attributed to the type of treatment advocated. It is conceivable that no single treatment can become standardized when various organisms are involved.

3. Suitably controlled animal inoculation experiments to ascertain the pathogenicity of all such cultures, bacterial and fungous, and an effort at better characterization of the relationship between the organism and the tissues.

4. Physiologic studies of pure cultures of the organisms with a view to possible substances that may be responsible for the symptoms or to other factors that would account for the pathologic changes observed.

Department of Biology, Vanderbilt University.

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OTOGENIC ABSCESS OF THE CONTRALATERAL FRONTAL LOBE

With Report of a Case

JULES G. WALTNER, M.D.
NEW YORK

THE FRONTAL lobe seems to be an uncommon site of otogenic abscess of the brain. Nielsen and Courville[1] made a most thorough survey of the literature and collected altogether 39 cases. In only 10 of these was the abscess a complication of otitis media occurring on the opposite side. No cases of contralateral otogenic abscess of the frontal lobe were disclosed by the records of the Presbyterian Hospital, New York. In 4 of the cases reported in the literature a chronic, and in 6 an acute, otitis media was the primary cause. Multiple abscesses were found in the opposite frontal lobe in only 1 of these cases. Thrombophlebitis of the venous sinuses on the side of the otitis was noted in 2 cases. In 6 of these 10 cases the diagnosis of abscess of the contralateral frontal lobe was first made at autopsy. In 8 of these 10 cases the abscess was actually found at autopsy. Two patients recovered after drainage of the brain abscess. The case presented in this paper showed a number of unusual features from the standpoint of the symptomatology and the development of acute abscesses of the brain in general and represents the third recorded case of otogenic abscess of the contralateral frontal lobe in which the patient completely recovered.

REPORT OF CASE

A 54 year old white woman was first seen the second day of earache on the right side, which was preceded by a mild infection of the upper respiratory tract. The right external auditory canal and drum membrane were partially covered with dark hemorrhagic bullas, some of which burst during examination, expelling a serous discharge. There was moderate tenderness over the mastoid process. Her temperature was 101 F. During the following ten days there was satisfactory drainage of purulent secretion from a posteroinferior perforation. No chills were observed. On the twelfth day of the otitis media the patient sud-

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From the Department of Otolaryngology of Columbia University College of Physicians and Surgeons, and the Presbyterian Hospital.

1. Nielsen, J. M., and Courville, C. B.: Otogenous Abscess of the Frontal Lobe: Review of the Literature and Report of Five Cases, Acta oto-laryng., 1937, supp. 21, p. 5.

denly noticed numbness of her right leg and arm and inability to move them, all of which occurred without any warning sign and completely surprised her while she was standing a few feet away from her bed. On being called to the patient's home, I found that she was suffering from complete paralysis of the flaccid type of the right leg and arm, although some motion of the fingers was still present. Nuchal rigidity was present. Her sensorium was clear. Her temperature was 103 F. The right ear was freely discharging; the left one was normal from every point of view. There were no signs or symptoms of sinusitis.

The patient was immediately taken to the Presbyterian Hospital, New York, where a spinal tap was done. The cerebrospinal fluid pressure was 280 mm. of water; there were 350 polymorphonuclear leukocytes and 170 lymphocytes per cubic millimeter of the fluid. The sugar content was normal. Smears and culture of the fluid revealed no organisms. The patient was given 5 Gm. of sodium sulfadiazine intravenously, followed by 1 Gm. orally, every four hours.

On the thirteenth day of the disease neurologic examination showed flaccid paralysis of the right leg and spastic paralysis of the right arm except for some motion of the fingers. The deep reflexes were weak on the right side. There was no Babinski reflex. The facial and the ocular muscles were unaffected. The eyegrounds were not remarkable. Kernig's sign was positive. The clinical impression of the neurologic consultant was: embolism of the middle cerebral artery of the left side. Spinal fluid obtained by a second tap revealed 660 leukocytes per cubic millimeter, 67 per cent of which were polymorphonuclears, and 136 mg. of protein and 28 mg. of sugar per hundred cubic centimeters. Intramuscular injection of penicillin was started, 20,000 units being given every three hours. The patient became progressively more somnolent, and her temperature rose to 105.4 F. Later in the afternoon she became incontinent. Culture of material taken from the right ear produced a growth of hemolytic streptococci.

On the fourteenth day the patient's sensorium became impaired and her nuchal rigidity more marked. There was some difficulty in swallowing, and motor aphasia was apparent. The paralysis of the upper right extremity extended to the right hand, all the motility of her fingers being lost. On the right there was present a Hoffmann, but no Babinski or other pyramidal signs. The cerebrospinal fluid pressure was 260 mm. of water, and 10 cc. of cloudy fluid was removed. A cell count showed 1,730 white cells, most of them polymorphonuclears. A blood count revealed 13,200 white cells, 95 per cent of which were neutrophils. Thirty thousand units of penicillin was injected intrathecally. The patient was seen in consultation by Dr. John D. Kernan, who suggested postponement of mastoidectomy if cultures of the spinal fluid remained sterile.

On the fifteenth day the patient became more alert, her nuchal rigidity decreased and her temperature dropped to 102 F. Again 30,000 units of penicillin was injected intrathecally. The patient was seen by the neurosurgical consultant, Dr. C. B. Masson, who found right hemiplegia with spasticity of the right upper extremity. No Babinski reflex was present on the right side. The abdominal reflexes were absent. The tendon reflexes of the right upper extremity were hyperactive; those of the right lower one, hypoactive. There was paresis of the right sixth cranial nerve. The right eyeground was essentially normal, while the left disk showed a slightly blurred edge but no elevation. Dr. Masson's impression was one of spreading encephalitis in view of the low cerebrospinal fluid pressure, the lack of papilledema and the spreading of the neurologic involvement. Continuation of the administration of the sulfonamide drugs and penicillin was advised.

One the sixteenth day the patient's condition improved. The rigidity of the neck definitely decreased, and the sensorium was much clearer. The aphasia was unchanged, but the patient promptly and correctly responded to orders. Her temperature steadily dropped. The cerebrospinal fluid pressure showed 270 mm., with a normal Queckenstedt reaction on the right side. Thirty thousand units of penicillin was injected intrathecally. There were 1,256 white cells in the spinal fluid, 56 per cent of which were polymorphonuclears. The spinal fluid protein amounted to 90 mg. and the sugar to 24 mg. per hundred cubic centimeters. All cultures of the spinal fluid were sterile.

On the seventeenth day the patient's condition showed no essential change. Hyperactive tendon reflexes were noted in both the upper and the lower extremities. The paralytic position was that of flexion of the right arm and extension of the right leg. Still present were spasticity of the arm and flaccidity of the leg. There was no Babinski reflex. Papilledema was not noted on the left. In the right ear there was copious mucopurulent discharge. The maximal temperature was 101 F. There were 1,120 white cells in the spinal fluid, 46 per cent of which were polymorphonuclears and 54 per cent lymphocytes. Thirty thousand units of penicillin was given intrathecally, in addition to penicillin intramuscularly.

On the eighteenth day the patient became more alert and the rigidity of the neck less obvious. The right hemiplegia was unchanged. The cerebrospinal fluid pressure was 150 mm. The cell count showed 950 white cells, of which 75 per cent were polymorphonuclears and 22 per cent lymphocytes. On the following day the patient started to talk and read without difficulty. The stiffness of her neck diminished, and her temperature dropped to 99 F.

On the twentieth and the twenty-first day further improvement was noted in the patient's speech and reading. There was minimal rigidity of the neck. Some motion returned to the right hand and fingers. Incontinence ceased.

On the twenty-fifth day motion of the fingers was again lost; however, speech remained normal. From a kidney-shaped perforation of the right drum membrane, a profuse, almost watery discharge was oozing. The perforation seemed to grow progressively. There was marked tenderness over the mastoid process. The spinal fluid showed 663 white cells, 59 per cent of which were polymorphonuclears and 34 per cent lymphocytes. The protein content was 136 mg. and the sugar 28 mg. per hundred cubic centimeters. No growth was observed on culture. Twenty-five thousand units of penicillin was injected intrathecally.

During the next four days the temperature rose slowly to 101 F., but there was no further development in the neurologic picture. On the twenty-eighth day the spinal fluid contained 102 white cells; the protein content was found to be 200 mg. and the sugar 41 mg.

On the thirtieth day of the purulent otitis media a profuse pulsating purulent discharge was coming through a large perforation in the drum. There was marked tenderness over the mastoid process, and roentgen studies of the right mastoid process revealed extensive destruction of bone. Simple mastoidectomy was performed, with the patient under ether anesthesia. Dental burrs were used in removing the cortex in order to avoid jarring of the skull. The superficial cells were hardly inflamed, while the deep retrofacial, perisinal and sinodural cells were extensively destroyed. Several large abscesses were discovered just anterior to the lowermost portion of the sigmoid sinus. The sinus was covered by granulation tissue from the region of the jugular bulb upward to an extent of 30 mm. Free blood was obtained from the sigmoid sinus on repeated

aspiration. The cerebral and cerebellar dura was extensively exposed and appeared to be normal. Cultures of material from the mastoid cells and of blood from the sigmoid sinus remained sterile.

Operation had no ill effect on the neurologic condition. The right hemiplegia was unchanged, and all meningeal signs disappeared. During the two weeks following the mastoid operation the infection of the middle ear cleared up and the operative cavity slowly filled in with healthy granulation tissue. At this time the administration of penicillin was stopped, and it was felt safe to take a pneumoencephalogram. The patient was transferred to the Neurological Institute, to the care of Dr. C. B. Masson. Air studies showed an expanding intracranial lesion on the left side, located fairly high in the region of the central fissure.

On the fifty-fifth day following the onset of the otitis media, left frontoparietal osteoplastic craniotomy was performed by Dr. Masson, with the patient under local anesthesia. The dura was normal in appearance. In the region of the motor strip a gyrus corresponding to the arm area was noted, which was flattened, larger than usual and dark in color in its uppermost portion. The arachnoidea was thin, and one of the vessels running into this area was probably thrombosed, being flanked along its course by a yellow, thick material. An abscess cavity measuring 3 by 2 by 2 cm. was found 5 mm. below the surface and was drained through a cortical incision 10 mm. in length. Sulfadiazine powder was put into the cavity, and a thin catheter was left in place for instillations of penicillin. Cultures of material from the abscess proved sterile.

After operation 30,000 units of penicillin was injected into the abscess cavity daily. The patient started to move her right hand two days after the operation, and slowly power returned to the arm and the thigh. She was able to walk unassisted and recovered full use of the upper extremity seventeen days after the drainage of the abscess. She was last seen fifteen months after her discharge from the hospital and has been free of complications.

COMMENT

Otogenic abscess of the contralateral frontal lobe is rare. The otologist is accustomed to search for an ipsilateral abscess of the temporal lobe or of the cerebellum occurring as a direct extension of suppuration from the temporal bone. Diagnosis of an otogenic abscess of the brain located in other than these common sites presents a difficult problem. In the present case there were a number of atypical signs and symptoms even for an abscess of the contralateral frontal lobe. The initial or invasion stage of an abscess of the brain, which is usually characterized by generalized or jacksonian seizures, severe headaches, slow cerebration, apathy, confusion and breathlessness, was absent in this case. The patient directly entered into the second or manifest stage, an apopleptic form of hemiplegia developing on the side of the otitis media. Eagleton [2] expressed the belief that sudden paralysis of the arm is pathognomonic of a large abscess of the frontal lobe. By reason of the experience gained from this case one has to

2. Eagleton, W. P.: Brain Abscess, New York, The Macmillan Company, 1922.

add that a sudden hemiplegia may occur in a patient who has an abscess of small size in the frontal lobe. It seems to be obvious that the location and not the size of the abscess will decide the nature of the symptoms. The frontal lobe covers a large area of the brain, and most of it is silent.

Meningeal signs seem to be uncommon in patients with otogenic abscess of the frontal lobe. Only 2 patients of those whose cases are recorded in the literature showed signs of meningitis. In the present case all the classic signs of severe meningeal involvement except a positive bacteriologic culture were present. The spinal fluid cell count was high, the globulin content increased, the sugar content decreased and the fluid pressure elevated. The sudden onset and spread of the abscess may explain the more marked involvement of the adjacent arachnoidal tissues. The aforementioned signs of meningitis do not necessarily mean meningitis in the pathologic sense. A meningeal irritation occurring in a circumscribed area over an abscess of the brain explains the actual condition. Therefore even if meningeal signs dominate the clinical picture, the possibility of an underlying abscess of the brain as the primary lesion should not be forgotten.

Aphasia followed the hemiplegia by two days. This was a great help in localization if not in identification of the pathologic process. The high fever at the onset of the hemiplegia was another confusing factor. According to many authors, high fever speaks in favor of encephalitis rather than an abscess of the brain.

In diagnosis it was of paramount importance to rule out otitis media of the left side or sinus disease as the possible source of the primary pathologic process. Examination of the chest revealed nothing which could possibly have been the primary source of metastases occurring in the brain. A pseudoabscess of the brain was another possibility, which could be ruled out only by further observation of the patient. A pseudoabscess is essentially a nonsuppurative, sometimes hemorrhagic encephalitis which either clears up spontaneously or shows regressions and recrudescences. In the course of acute mastoiditis transient paralysis and convulsions are more frequent in young children. Embolism of the middle cerebral artery associated with meningeal signs is another condition which could have explained the patient's sudden hemiplegia during the early stage of the complication.

The therapeutic handling of the patient presented a most difficult problem during the first three days of the hemiplegia, owing to the fact that an accurate diagnosis of the lesion present in the brain was impossible during the first stages of the hemiplegia. The patient was in a desperate condition, and the cerebral lesion seemed to be spreading (first hemiplegia, then aphasia and incontinence). Although there was no doubt that the right-sided acute purulent otitis media was a definite,

and most probably the only, focus of infection in the body, it was decided to postpone the eradication of the focus, because little could be expected in the way of clearing up the hemiplegia from mastoidectomy. On the other hand, the possibility of a sudden spread of involvement of the brain due to a jarring of the head and stirring up of the focus weighed heavily in favor of conservative treatment. The combination of the use of sulfadiazine and the intrathecal and intramuscular injection of penicillin cleared up the meningeal involvement but not the mastoiditis, and thus gave time for longer observation and encapsulation of the abscess of the brain. The prognosis thus improved every day. Early drainage of brain abscesses gives notoriously poor results from the point of view of survival and function.

It is noteworthy that continuous combined therapy with sulfadiazine and penicillin over a period of seventeen days did not prevent extensive destruction of the deep cells of the mastoid process, while the superficially located ones were hardly diseased. This may well be one of the explanations for disappearance of tenderness over the mastoid process on administration of penicillin and sulfadiazine, which may lead to an unwarranted optimistic prognosis.

The nature of the brain lesion was definitely established by air studies on the thirtieth day of the hemiplegia, and, following this, the encapsulated abscess of the frontal lobe was successfully drained through the left frontoparietal osteoplastic craniotomy.

The route by which the infection spread from the right mastoid process into the left frontal lobe of the brain is hard to determine. In most cases described in the literature, even serial sections of the brain did not enable the observer to trace the route of infection. Courville and Nielsen [1] tried to explain the formation of distant otogenic abscesses of the brain by a retrograde venous spread following primary thrombophlebitis of the sigmoid sinus. The present case seems to give support to this explanation. Extensive phlebitis, if not thrombosis, was found in the lower half of the sigmoid sinus, and a thrombosed vessel was found over the area of the brain which lay just above the abscess. The superficial location of the abscess speaks against an arterial metastatic spread. Truly metastatic abscesses are more likely to occur in the center of the brain, where the blood supply is the poorest.

The high mortality caused by contralateral otogenic brain abscesses is not uniquely due to diagnostic difficulties, although most of those described in the literature were diagnosed at autopsy. The occurrence of multiple abscesses adds to the therapeutic and diagnostic problems. The poor prognosis of these abscesses is only mildly mitigated by the fact that a diffuse suppurative type of encephalitis was not observed in any of these patients. The abscess of the temporal lobe which occurs as a complication of cholesteatoma of the middle ear more frequently goes through a stage of diffuse suppurative encephalitis.

In the eventual complete recovery of the present patient, especially from the point of view of function of the extremities, the timing of the mastoidectomy and the operation on the brain seems to have been of paramount importance. Postponement of the eradication of the obvious mastoiditis was imperative in the light of the aforesaid operative risks. Spreading of the abscess of the brain might have occurred, but not necessarily, without the administration of sulfadiazine and penicillin.

SUMMARY

A case of acute purulent otitis media complicated by abscess of the contralateral frontal lobe is presented as the third one to be recorded in the literature in which the patient showed complete recovery.

Hemiplegia occurring suddenly on the side of the otitis media is to be added to the symptoms of otogenic abscess of the frontal lobe. This may be the very first sign of involvement of the brain.

Both the possibility of a severe meningeal reaction occurring over the abscess of the brain and the possibility of a so-called pseudoabscess make the immediate diagnosis of a contralateral otogenic abscess of the brain frequently impossible. In most instances reported in the literature the abscess was found only at autopsy.

The present case supports the theory of a retrograde venous spread of thrombophlebitis of the sigmoid and lateral sinuses.

The eradication of the focus of infection discovered in cells of the mastoid process and the drainage of the abscess of the brain after waiting long enough for encapsulation of the abscess, combined with the administration of sulfadiazine and penicillin, resulted in a complete cure of the patient.

630 West One Hundred and Sixty-Eighth Street.

ENDOSCOPIC USE OF THE BIPLANE FLUOROSCOPE

E. W. HAGENS, M.D.
CHICAGO

WHILE the biplane fluoroscope is not needed in the majority of cases of foreign body, nevertheless there are instances in which its use is either extremely helpful or absolutely necessary. It has been my fortunate experience to have worked in a hospital where since 1931 the biplane fluoroscope has been available (Presbyterian Hospital, and Wesley Hospital since 1942). Because of its convenience, the rule was made that the biplane fluoroscope was to be used on most opaque foreign bodies. As a result there were many cases in which the biplane fluoroscope proved effective. In the majority of instances the fluoroscope was not actually necessary, but in some cases it was valuable when something went amiss. The use of the fluoroscope was not in any way a substitute for the usual technic but was only an added safeguard. Of the group of cases in which the biplane fluoroscope was of aid, 7 have been chosen to illustrate the value of the instrument.

REPORT OF CASES

CASE 1.—Joan C., aged 2, swallowed a penny three days before coming to the hospital. Roentgenograms showed the penny in the usual place in the upper part of the esophagus. Esophagoscopy was started without the aid of the biplane fluoroscope, and though a thorough search was made, especially on the posterior wall just below the cricopharyngeus muscle, no evidence of the penny could be found. The child was taken to the x-ray department, where single plane fluoroscopy showed the penny still present. The patient was then placed on the biplane fluoroscope and the esophagoscope inserted just to the lower end of the cricopharyngeus muscle. The roentgenologist reported that the coin was anterior to the tube. By pointing the esophagoscope strongly anteriorly the penny was seen hugging the anterior wall of the esophagus. After several attempts to grasp the coin it was found necessary to dislodge the coin with the lip of the tube, and by holding it there the penny was finally grasped with side-grasping forceps and removed.

Comment.—The penny was so flattened against the anterior wall of the esophagus that it was not seen until the biplane fluoroscope revealed its site.

CASE 2.—Mrs. X, aged 35, was seen in the medical department for general symptoms. On roentgenographic examination of the chest a needle was found

Presented as a candidate's thesis to the American Broncho-Esophagological Association.

which was apparently in the dorsal branch of the bronchus of the lower lobe of the right lung. The patient gave no history whatever of inhaling the foreign body, but apparently it had been present a long time. Several attempts had been made elsewhere to remove the needle, but as it could not be located the patient was referred for biplane fluoroscopy and endoscopy. With the aid of the fluoroscope I was able to find the site of the pin, inserting the lips of the side-grasping forceps into the mouth of the dorsal bronchus. Attempts to grasp the foreign body showed proper alinement in the two planes and movement of the needle. However, no metallic feel was imparted to the forceps tip, and apparently the needle was embedded in the bronchial tissue. After grasping a number of times and being unable actually to touch the object, the effort was given up. The patient failed to report later as she had been advised to do, and nothing further was heard from her.

Comment.—Although the buried needle could not be removed, the biplane fluoroscope made it possible to localize and find the site of the foreign body, even to the point of moving it.

CASE 3.—Mr. M., aged 42, a carpenter, accidentally aspirated a 3 penny nail. Roentgenograms showed the object, point up, in the right lung. For years the patient had suffered from bronchitis with moderate secretion, but since the foreign body was inhaled the cough had been worse. Removal was attempted, the biplane fluoroscope being used. The nail was in the lower lobe bronchus and its upper part could be seen through the bronchoscope. On grasping it, however, owing to bloody secretion and the excursions of the bronchus, the nail was caught too low on its shaft so that the point dug into the bronchial wall when an attempt was made to pull it up. By following the instructions of the roentgenologist the tip of the nail was grasped and the nail easily removed. Uneventful recovery occurred.

Comment.—As in this case, when blood, secretions and respiratory movements combine to make visualization of the foreign body difficult the biplane fluoroscope is helpful in properly grasping the object.

CASE 4.—Mr. F., aged 52, a carpenter, inhaled a nail (brad) into the right lung. An endoscopist's attempt to remove the nail, working without aid of the biplane fluoroscope, failed after an hour's search. The patient was referred for removal of the nail under fluoroscopic guidance. Roentgen and fluoroscopic examination showed the brad to be in the bronchus of the lower lobe of the right lung with the head near the diaphragm. With the use of the 5 by 45 bronchoscope and with the help of the biplane fluoroscope the nail was found and easily removed in three minutes.

Comment.—The biplane fluoroscope changes a difficult case into a rather simple one.

CASE 5.—Harold B., aged 10, inhaled a metal wing of a toy airplane two months before being seen. He coughed for some weeks and finally was taken to the hospital with what was thought to be pneumonia. Roentgenograms revealed the foreign body in the left lung, and the true history was then obtained from the patient for the first time. When the acute pulmonary reaction subsided, the boy was transferred to me for fluoroscopic removal. When the bronchoscope was inserted into the left main bronchus a collar of granulation tissue was found midway down the bronchus. This so narrowed the bronchus that the tube could not

be passed through it, nor could the foreign body be seen. Evidently the prolonged constant coughing had stimulated this tissue to develop in the bronchial mucosa just where the upper end of the metal touched the mucous membrane. Bearing in mind the plane of the metal wing as studied on the anteroposterior and lateral roentgenograms and being guided by the roentgenologist controlling the biplane fluoroscope, I was able to grasp the object correctly with the forward grasping forceps and to remove it. Recovery occurred rapidly, the incessant coughing practically ceasing in twenty-four hours.

Comment.—Without the aid of the biplane fluoroscope it might have been quite a task to find the proper plane in which to apply the forceps to the foreign body, as it could not be seen, owing to the proximal "collar" of granulation tissue.

CASE 6.—Miss O., aged 17, complained of a cough which had developed nine years previously. For most of this time she had taken various types of cough medicine. For the last three years there was expectoration in the morning. Recently the girl had undergone pneumonia and empyema. It was while studying the roentgenograms of the chest that the presence of a three-fourths inch safety pin was noted in the right lung. The patient then admitted that nine years before she had "choked" on the pin but had never mentioned it to any one. At bronchoscopy, using the biplane fluoroscope, I found the pin, point up, in the right main bronchus. While it was corroded, there was little else of note, and the pin was grasped with the keeper and straightened into the tube mouth. Examination of the safety pin on removal showed that the point had been lost, and the patient was immediately studied with the biplane fluoroscope. The point was not in the lung and was soon found in the stomach, the corroded part evidently having broken off in the pharynx and passed down the esophagus.

Comment.—The biplane fluoroscope made it possible in this case to locate the broken point of the safety pin, so the reexamination of the bronchial passages was unnecessary.

CASE 7.—Marc E., aged 2, swallowed an open safety pin twenty-four hours previously, and roentgenograms showed the pin, point up, opposite the sixth to eighth dorsal vertebrae. Under fluoroscopic guidance the head of the safety pin was grasped and an attempt made to straighten the pin into the tube. However, the keeper broke off, and this alone came out with the forceps. Study with the fluoroscope showed the safety pin in the original position but minus the keeper. With fluoroscopic guidance the pin was grasped at the keeper end once more, straightened into the tube, and removed.

Comment.—In this case the pulling off of the keeper of the safety pin immediately precipitated the need for fluoroscopic aid.

CONCLUSIONS

It is evident that when it is possible to have the help of the biplane fluoroscope the endoscopist can do safer and better work than otherwise. It is to be used as an aid in bronchoscopy, and not as a substitute

for the usual proper technic. The biplane fluoroscope helps (*a*) to localize a foreign body when it can otherwise not be found, (*b*) to furnish accurate and immediate information when the foreign body changes position rapidly or comes apart or breaks, (*c*) to determine whether the grasp on the foreign body is proper or not, and (*d*) to find the new position of an object which has suddenly disappeared from view, thus obviating unnecessary roentgen examination and manipulation.

30 North Michigan Avenue.

THE VESTIBULAR APPARATUS AS AN ORGAN OF SENSE

ERNEST WODAK, M.D.
TEL-AVIV, PALESTINE

IT IS a striking fact, as I explained in more detail in another place,[1] that one encounters the same signs, reactions and other phenomena in the examination of the pathologic vestibular apparatus as one is accustomed to observe in the examination of the normal apparatus—but exaggerated, sometimes even excessive. It is quite understandable that one does not always find in a single case all the known vestibular phenomena, e. g., tonic body reflexes, nystagmus and dizziness, at the same time. Usually one or more of these symptoms is the main feature of the disease.

From these facts one may conclude, first, that when one examines the vestibular apparatus of the normal person, all the extant phenomena, such as vertigo, body reflexes and nystagmus, endanger the equilibrium and are normally and physiologically quickly suppressed. Every normal person has in his central nervous system some physiologic checking mechanism which protects the endangered equilibrium either by unconscious or by partly conscious movements and changes of position. One may assume not only one checking mechanism for each of the mentioned vestibular reactions but probably, for safety, several such mechanisms for each vestibular phenomenon. One such protective mechanism may be in the frontal lobe; another, according to Alexander Spitzer, in the Bechterew commissure, and, perhaps in addition to these, another may be in the cerebellum. If one or more of these checking mechanisms are disturbed or put out of order by some pathologic process, the vestibular phenomena concerned, otherwise checked and controlled, are released (*enthemmt*) and dominate the clinical picture of the disease.

The second conclusion that one may make is as follows: The vestibular phenomena which one observes in the examination of normal persons are not physiologic, but pathologic. They do not, however, impress one as pathologic phenomena because in normal persons they are rather insignificant and, furthermore, immediately stopped by the protective mechanisms mentioned. Therefore, the usual examination of

1. Wodak, E.: Vestibular Apparatus and Neurology, Acta oto-laryng. orient. **2**:10, 1946; Some Remarks as to the Physio-Pathology of the Vestibular Apparatus, Acta oto-laryng., **34**:593, 1946.

the vestibular apparatus of the normal person, if its result is normal, proves: (*a*) that the peripheral vestibular apparatus itself, together with the vestibular nerve and its central nucleus as well as the proceeding pathways (ductus deiterospinalis, fasciculus longitudinalis posterior) and the hypothetic pathway of the vertigo, is intact and its function normal; (*b*) that the physiologic apparatus which checks and controls these vestibular phenomena is in working order, since these phenomena are stopped immediately. One may call only this checking mechanism a physiologic one whereas, as mentioned, the insignificant vestibular phenomena observed in normal persons have to be considered pathologic. Therefore, the usual examination of the vestibular apparatus is not of the same kind as that of other organs of sense, such as the eye or the ear, but is something like a test of strain (*Belastungsprobe*).[2] By the examination of the vestibular apparatus one attempts to determine whether by adequate stimulation (turning test) or by inadequate stimulation (caloric test, galvanization), one is able to evoke the typical vestibular phenomena, all of which endanger the vital equilibrium. Furthermore, one wants to know whether the physiologic checking mechanism works properly.

The theoretic superposition of this restraining mechanism enables one to advance in diagnosis. One can now, perhaps, distinguish between peripheral and central hyperexcitability of the vestibular apparatus. Peripheral hyperexcitability may be assumed if the latent period between the unilateral caloric stimulation and the manifestation of the vestibular phenomena is much shorter than it would be normally. This latent period is normally about fifteen to twenty seconds when one uses minimal calorization (15 to 20 cc. of cold water). One is entitled to make the diagnosis of hyperexcitability when this period is about five to eight seconds or less. If the manifest vestibular symptoms, such as tonic body reflexes, nystagmus and vertigo, are not stronger than they usually are, one may assume that there is moderate peripheral hyperexcitability. If, however, these symptoms are by far stronger than those normally occurring but do not last longer than the usual reaction, one may speak of a higher degree of hyperexcitability. On the other hand, if the latent period is unchanged, but the vestibular phenomena last much longer than those of normal persons (three minutes or more), one may then assume that there is central hyperexcitability. Naturally, one has to be most cautious in one's judgment; only an impressive shortening of the latent period as well as a lengthening of the reactions should be noted and considered.

2. One knows in internal medicine the analogous tests of strain usually employed in the examination of the liver, the kidneys and other structures.

One must remember that years ago Alexander and Brunner [3] distinguished between peripheral hyperexcitability and central hypersensibility of the vestibular apparatus. Their criteria were almost the same (of course, without the interpolation of a checking mechanism) as mine. We came to the same result, only in different ways.[4]

Years ago I tried to perfect a method of utilizing the dissociation of the different vestibular reactions for diagnostic purposes.[5] Then I distinguished but one kind of hyperexcitability and noted a difference only if there was hyperexcitability for all vestibular reactions rather than for one or another. Now, with the definition of peripheral and central hyperexcitability, one has to enlarge the general idea of hyperexcitability and make some subdivisions for diagnostic purposes. One may thus gain some diagnostic hints.

This leads one to the problem of the vestibular apparatus as an organ of sense and its behavior, compared with other organs of sense. Comparing it with the visual or the acoustic organ (the organ of smell or that of taste may show similar qualities) one can state:

1. Stimulations, i. e., wavelengths, below the physiologic threshold which meet the retina or the cochlea do not cause any perception. The same holds for stimulations below the threshold which reach the vestibular organ.

2. Stimulations above the threshold but still between the physiologic boundaries which reach the retina or the cochlea and proceed on certain central pathways to the visual or the acoustic sphere will be transformed there into optic or acoustic perceptions and so will come to consciousness. This is not true for the vestibular organ; stimulations of this physiologic kind do not cause any perception which reaches consciousness.

3. Stimulations not only above the threshold but beyond the physiologic boundaries cause visual or auditory pathologic reactions: pain, reflex closure of the eyelids or instinctive movements to protect the endangered organ. The stimulations of the vestibular organ, even in the usual examination of normal persons, are always of this kind and therefore pathologic ones. Thus one must also consider reactions

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3. Alexander, G., and Brunner, H.: Ueber labyrinthäre Ueberregbarkeit, Ztschr. f. Hals-, Nasen- u. Ohrenh. **3**:243-257, 1922.

4. C. A. Elsberg in his article "Localisation of Tumors of the Brain by Means of Tests by Olfactory Function" (A. Research Nerv. & Ment. Dis., Proc. [1935] **16**:327, 1937) made a similar distinction between peripheral and central disturbances of the sense of smell. He assumed a peripheral disturbance when the latent period was shortened, whereas prolongation of the fatigue period (more than in normal patients) spoke for a central disturbance.

5. Wodak, E.: Exact Clinical Differentiation of Conditions of Excitability of Vestibular Apparatus, Acta oto-laryng. **9**:245-253, 1926.

such as sensation of rotation, tonic body reflexes, nystagmus and vertigo as pathologic phenomena which endanger vital equilibrium. In consequence, the stimulated or irritated vestibular organ sends certain signals of warning to the center which, for their part, induce the aforementioned checking mechanism to intervene and to protect the endangered equilibrium. What is lacking in the behavior of the vestibular organ, and this distinguishes it from the other organs of sense, is that it does not answer the stimulations which are above the threshold but still between the physiologic boundaries with a conscious reaction as the other organs do. More precisely, one is not conscious of a vestibular response to such stimulations, if there is any.

The organs of touch, especially those of deep sensation (*Tiefensensibilität*), are similar in their working to the vestibular organ but different from the eye, the ear and some others. Here again one is not conscious of stimulations above the threshold but between the physiologic boundaries. One is conscious only if excessive stimulation is present; e. g., when one is sitting or standing on an inclined plane, so that one's natural erect position is disturbed (A. Tschermak), one becomes conscious of this disturbance and makes either conscious or unconscious movements to restore the vertical position. Therefore, of all the organs of sense, only the vestibular organ and those of deep sensibility work in a similar manner, thus contrasting with the other organs of sense, including those of smell and taste. It is interesting to note that both of these organs, working toward the same objective, play essential, though not similar, roles in the preservation of the body's equilibrium.

ANESTHESIA FOR THE SURGICAL CORRECTION OF DEAFNESS

S. G. HERSHEY, M.D.
AND
MARVIN F. JONES, M.D.
NEW YORK

FENESTRATION for the surgical improvement of deafness was introduced in this country by Lempert in 1938.[1] Since then, in the past five years particularly, the number of patients undergoing this operation has grown rapidly. It is certain that many more will seek surgical intervention for otosclerosis in the immediate future, since fenestration is gaining popularity, and an increasing number of otologists are performing this painstaking and delicate procedure.[2]

The fenestration operation is, perhaps, the most delicate surgical procedure performed on human patients. With the development of this new operation it becomes the function of the anesthetist to provide an anesthetic regimen for it which is both safe for the patient and adequate to meet the surgeon's technical requirements. To do this, the anesthetist should become familiar with the essential principle of the operation and the steps in the technic of its completion.[1]

SURGICAL AND ANESTHETIC PROBLEMS

The problems directly related to anesthesia which such surgical practice has created for the surgeon are few but exacting. The main deterrent which diminishes the percentage of satisfactory results is bleeding, which varies with the anesthetic drug and the technic. Next in importance is the maintenance of light anesthesia which can be safely augmented at such times as the pain stimulus is increased, since complete immobility is absolutely necessary at these times. It is also reassuring and of distinct advantage to have the patient in a cooperative state immediately after the operation.

From the Department of Anesthesia of New York University College of Medicine, Beth Israel Hospital and the Otolaryngology Service of the Manhattan Eye, Ear and Throat Hospital.

1. Lempert, J.: Improvement of Hearing in Cases of Otosclerosis: A New One Stage Surgical Technic, Arch. Otolaryng. **28**:42-97 (July) 1938.

2. Shambaugh, G. E., Jr.: The Fenestration Operation for Otosclerosis, J. A. M. A. **130**:999-1006 (April 13) 1946. Martin, R. C.: Fenestration Operation for Deafness, California & West. Med. **62**:1-6 (June) 1945.

In the initial operative phase, viz., incision, bleeding causes an annoying delay but has no deleterious effect on the final result. A hemostatic can be utilized. During the second phase, removal of the mastoid cortex and exenteration of pneumatic cells, a bloodless field markedly reduces the time spent. In the next phase the procedures all involve sensitive structures of the middle ear and the external auditory canal. Annoying bleeding, difficult to control, may follow exaggerated manipulation of the membranous external auditory canal as a result of the patient's straining. Bleeding and straining both affect the final result, since the structures in the area are extremely important and must be carefully preserved. For the final phase, making the fenestra, the patient must be quiet, and bleeding must be completely controlled.

From the anesthetist's point of view the following details of the surgical problem and technic enter into the conduct of anesthesia. Most surgeons use electrocoagulative current to control soft tissue bleeding. It is also almost mandatory that a motor-driven dental burr be employed to prepare the sclerotic mastoid cavity and define the semicircular canal. This electrical apparatus in close proximity to the respiratory passages automatically eliminates the flammable inhalant drugs as anesthetic agents.

For most of the procedure very light anesthesia is sufficient. Though the depth of narcosis need never be profound, there are several intervals in which operative stimulation is increased so as to require a surgical plane of anesthesia. These intervals occur with the incision, the separating of the periosteum from the mastoid bone, again whenever the dental burr or the curets are used close to or in the cavity of the middle ear and when the tissue flap is cut from the wall of the auditory canal. Another period of increased stimulation, of a different nature, occurs at the close of operation when hot liquid paraffin is poured into the mastoid cavity and this vigorously irrigated with iced saline solution. The direct application of heat and cold to the vestibular system will evoke a reflex vomiting response unless the patient is in surgical anesthesia. To accommodate for these, anesthesia must be maintained with sufficient flexibility so as to permit for rapid changes in depth.

For the surgeon, bleeding is, as previously mentioned, the greatest source of interference. The bleeding referred to is neither arterial nor capillary but of venous type, from the haversian systems of the bone. Anesthesia must of necessity be conducted without drugs or agents which of themselves or by their method of administration will cause increased bleeding.

ANESTHETIC MANAGEMENT

Premedication.—Pentobarbital sodium, 0.1 Gm., is administered orally two hours preoperatively. Morphine sulfate, 0.01 Gm., and scopolamine hydrobromide, 0.0004 Gm., are given hypodermically ninety minutes before operation. Smaller doses of these drugs are utilized in older patients.

Basal Narcosis.—Solution of tribromoethanol U. S. P. (80 to 90 mg. of tribromoethanol [40 to 45 mg. of amylene hydrate] per kilogram of body weight) as a 3 per cent solution in distilled water is instilled rectally thirty minutes before scheduled time. To decrease the toxicity of tribromoethanol in relation to the liver, the two meals preceding operation are ordered to be rich in carbohydrate and a saline-dextrose infusion is always given immediately after operation. In relation to the possible development of respiratory depression from solution of tribromoethanol U. S. P. and morphine premedication, the patient is brought to the operating room as soon as he becomes drowsy, so that oxygen can be administered well before the full effect of basal narcosis occurs.

Inhalation Anesthesia.—Nitrous oxide is administered throughout, a semiclosed absorption to and fro technic being used. The concentration varies from 50 to 75 per cent, according to the depth of anesthesia required. The concentrations of nitrous oxide delivered are carefully measured on a gas machine which has a flowmeter specially calibrated for small volumes of this gas. Scrupulous attention to the delivered concentrations of gas to eliminate the hypoxic range is particularly important, since the operation is performed in a darkened room and the patient's color cannot be used as a guide to oxygenation.

The evidence of increased stimulation requiring an increase in the concentration of nitrous oxide consists in augmented rate and depth of respiration. When these signs continue with concentrations of nitrous oxide of 75 per cent, rather than increase this, give morphine sulfate, 0.01 Gm., intravenously. This additional dose is necessary only occasionally and has never been required until operation has been under way for ninety minutes (three and one-half hours after initial pentobarbital, and three hours after morphine, premedication).

Airway.—In the operating room all patients are lightly cocainized by spraying a few cubic centimeters of a 4 per cent solution of cocaine hydrochloride into each nostril. Both an oropharyngeal and a nasopharyngeal airway are inserted very gently.

The procedures described plus the topical anesthesia and the favorable position on the operating table (head in slight extension and turned to the side) serve entirely well. The ordinary Connell face piece and mastoid head strap when carefully placed are well out of the surgical field.

COMMENT

Before the present clinical study was started the method of inducing anesthesia most often employed [3] consisted of an analgesic regimen supplemented by local procaine infiltration and, on occasion, administration of nitrous oxide in addition. In the two hours preceding operation the patients were given 0.2 to 0.4 Gm. of pentobarbital sodium or a comparable barbiturate, 0.015 to 0.030 Gm. of morphine sulfate in divided doses and a single dose of atropine sulfate (0.0003 Gm.). Nitrous oxide when needed was administered by a semiclosed technic. Procaine was used for the initial incision. The disadvantages of such heavy use of the depressant drugs and the relative lack of safety are generally conceded.

Our experience with the use of the outlined method of inducing anesthesia is only preliminary in nature. Although the clinical results seem

3. Shambaugh, G. E. Jr: Personal communication to the authors. Lempert.[1]

satisfactory to date, it would serve no purpose to present any statistical evaluation until many more records have accumulated. There is little doubt that increased experience will result in changes of this anesthetic management of patients undergoing fenestration. One objective of this preliminary report is to suggest that the more recent developments of clinical anesthesiology be more widely applied to this relatively new development of surgery.

Some aspects of the present method should be amplified in relation to the specific details of fenestration. Bleeding from the bony haversian system, previously referred to, assumes importance out of all proportion in this as compared with other operations. Anesthesia can either greatly impede or aid the surgeon in regard to a "bloodless" field. It cannot be well controlled by clamps, electrocoagulation, topical application of epinephrine hydrochloride or use of bone wax. Frequent or even continuous irrigation of the cavity is utilized. This bleeding literally is greatly magnified in the latter phase of the operation when the surgeon, to increase his vision, uses binocular dissecting microscope type objectives in creating the fistula. The anesthetist's responsibility in this regard is to avoid hypoxia and any degree of respiratory obstruction leading to increased systolic blood pressure and increased venous and intracranial pressure. If these conditions develop, they are reflected by increased bleeding not only for the transient period of their duration but for many minutes following it.

The selection of basal narcosis induced with solution of tribromoethanol U. S. P. is also related to the problem of bleeding. If the systolic blood pressure is somewhat depressed, less bleeding is expected. From experience with the depressor tests in selecting hypertensive patients for sympathectomy it is known that certain barbiturates and solution of tribromoethanol U. S. P. both depress the systolic blood pressure.[4] This effect can be anticipated in normal (nonhypertensive) patients to a lesser degree. In choosing solution of tribromoethanol U. S. P. its advantages were weighed against the intravenously administered dilute (0.2 per cent) solution of pentothal sodium. With the former the patient has no recollection of going to the operating room. Though pentothal sodium when injected in dilute solutions over long periods is probably entirely safe, it does not yet enjoy the generally accepted usage of solution of tribromoethanol U. S. P. and is a more potent respiratory depressant. Furthermore, in such dilute solutions pentothal sodium does not produce the fall in blood pressure with the regularity of solution of tribromoethanol U. S. P.

4. Phelps, M. L., and Burdick, D. L.: Anesthetic Management of Patients Undergoing Sympathectomy for Hypertension, Anesthesiology **4**:361-371 (July) 1943.

In several instances, early in this study, a nasotracheal catheter was inserted after the cocaine spray without the use of a laryngoscope. Though it insured a perfect airway, the use of an endotracheal catheter was abandoned because of the occasional coughing during operation which it produced in these lightly anesthetized patients. Such episodes increase bleeding both during and after their occurrence. The use of combined oronasopharyngeal airways clearly constitutes a compromise with ideal inhalation technic for surgical operations about the head. It is likely that further experience will bring a return to the endotracheal technic.

There are other considerations peculiar to the fenestration operation. As a group, these are well patients voluntarily seeking physiologic restitution of a nonvital function: hearing. Their experiences during surgical treatment can, to a considerable extent, influence the popularity of such a wholly elective operation. The anesthetist must share the responsibility of making the patient's experience as little unpleasant as is feasible, being mindful, however, that a serious complication can significantly dampen present enthusiasm. He can also share in the further development of this new operation by providing the surgeon with the working conditions needed to complete the procedure with maximum efficiency.

SUMMARY AND CONCLUSION

The surgical and anesthetic problems involved in the fenestration operation are outlined and an anesthetic regimen, preliminary in nature, is presented in relation to these problems. It is important to develop anesthetic care for this new operation according to current concepts of clinical anesthesia.

477 First Avenue.

TYROTHRICIN IN THE TREATMENT OF THE COMMON COLD

DUANE DARLING, M.D.
CHICAGO
AND
CARL F. BAUMEISTER, M.D.
RIVERSIDE, ILL.

OF ALL the annoying and nonfatal diseases which plague mankind, the most frequent is the common cold. That it is not entirely harmless must be recognized. It takes a tremendous economic toll each year, causing the loss of more man–hours among the workers of this country than any other disease. It is often the predecessor of far more serious ailments; notably pneumonia, sinusitis, bronchitis, otitis media and mastoiditis. If it does not confine people to bed, it accounts for a great deal of discomfort and a loss of considerable efficiency for at least two weeks out of the average man's year.

Much time and money have been expended in attempts to determine its cause. Countless remedies have been tried for its prevention and treatment. The net result of all these endeavors has been so unsatisfactory that the subject has long been a fertile field for cartoons and jokes at the expense of the medical profession. The tremendous sales of patent medicines for colds is mute evidence of the inadequacy of the therapeutic armamentarium.

MODERN TREATMENT OF HEAD COLDS

In general, most colds start in the upper respiratory tract, usually as rhinitis or pharyngitis. The condition may remain confined to this region, or it may start in, or spread to, the larynx, the bronchi and the lungs. In addition, all head colds produce some systemic symptoms which vary from mild to severe.

Treatment to date has been largely supportive, the doctor hopefully, if not apologetically, suggesting fluids, rest and warmth. Antipyretic drugs, codeine and papaverine are used to allay discomfort.

Interest in more specific therapy has waxed and waned. Ethical medicine has frowned on the extensive and credulous use of the so-called cold vaccines whether for prophylaxis or treatment. Vitamins, particularly large amounts of vitamin A, have fallen from popularity. The

sulfonamide drugs have proved of little value. A host of antiseptic agents, notably the recent mercurial derivatives, have been discarded.

It must be agreed that modern science does not have much to offer for the prevention or the treatment of the common cold.

USE OF TYROTHRICIN FOR HEAD COLDS

Because of the success with which we had used tyrothricin on lesions of skins and mucous membranes, it was decided to try its efficacy on membranes affected by the common cold. Beginning on Aug. 20, 1944, tyrothricin was used in various concentrations in different suspensory mediums in the treatment of head colds. The best suspensory medium was found to be one of the synthetic vasocontrictor nose drops.[1] It was determined that the optimum concentration was 0.5 cc. of 2 per cent tyrothricin in 14.5 cc. of suspensory medium. If weaker solutions of tyrothricin were used, insufficient antiseptic effect was obtained. With the use of stronger solutions there was attendant discomfort, including stinging and watering of the eyes and the nose.

The solution was instilled into the nasopharynx as often as every hour for the first few doses and every four or five hours thereafter. Then patients were advised to use it frequently enough to avoid, to a reasonable extent, the nasopharyngeal discomfort which those who have had colds know so well.

The effects of the solution were both local and systemic. The local effect consisted of a peculiar drying sensation not reproduced with other nose drops and contributing greatly to the patient's comfort. The systemic effect was apparent in a decrease of the toxic feeling so often accompanying a cold.

Since the potency of the mixture began to decrease after seven to ten days only ½ ounce (15 cc.) was prescribed, and the patients were advised to keep the bottle tightly capped and in a refrigerator. Giving only 15 cc. of solution on a nonrefillable prescription avoided untoward effects which might follow excessive use of the nose drops.

CORRELATION OF RESULTS

Three hundred and sixty persons in whom head colds were developing were treated with nose drops; 250 used the nose drops including tyrothricin; 110 (controls) used nose drops without tyrothricin.

No attempt was made to compare the peculiar drying effect produced in the nasopharynx by the tyrothricin nose drops, since this effect was not obtained with other solutions.

Table 1 shows a comparison made in different seasons of the plain and the tyrothricin nose drops with respect to the decrease of toxic effect achieved within six to eighteen hours. It is well recognized

1. A 1 per cent solution of para-hydroxy-alpha-methylphenethylamine hydrobromide.

that many head colds will subside spontaneously. However, there was a noticeable difference between the effects produced by the tyrothricin nose drops and the plain nose drops on the early fall and late spring head colds. In those colds occurring during the winter this difference was not so pronounced. Possible reasons for the marked seasonal variation in the effectiveness of the tyrothricin solution is not in the province of this paper. They will be discussed in future publications.

Table 2 shows a comparison of plain nose drops and those incorporating tyrothricin with regard to the percentages of complications. The most marked effect was a reduction of the incidence of sinusitis. With regard to all the complications there is some difficulty in interpreting

TABLE 1.—*Decrease of Complications*

	110 Cases in Which Plain Nose Drops Were Used			250 Cases in Which Nose Drops plus Tyrothricin Were Used		
	62 Cases		48 Cases	121 Cases		129 Cases
Complication	Early Fall	Late Spring	Winter	Early Fall	Late Spring	Winter
Otitis media (reddening drum; pain in ear)	5 cases 8%	No cases	8 cases 16.6%	6 cases 4.96%	....	17 cases 13.2%
Sinusitis (sinus headache; persistent nasal discharge)	17 cases 27.4%	....	16 cases 33.3%	20 cases 16.5%	....	25 cases 19.4%
Tracheobronchitis.....	30 cases 48.4%	....	31 cases 64.6%	45 cases 37.2%	....	62 cases 48%

TABLE 2.—*Decrease of Toxic Symptoms*

Nose Drops Used	Early Fall and Late Spring	Winter	Over All
Plain nose drops—110 cases	23 cases out of 62 37.1%	11 cases out of 48 22.9%	34 cases out of 110 30.9%
Nose drops plus tyrothricin—250 cases	79 cases out of 121 65.3%	33 cases out of 129 25.6%	112 cases out of 250 44.8%

the efficacy of the various drops, since some of the complications may be present in a subclinical state at the time of the inception of therapy.

SUMMARY

Tyrothricin suspended in a synthetic vasoconstrictor solution was found to be useful in the treatment of early head colds. The combination effected an appreciable diminution of toxicity, of duration and of complications of the common cold. Such beneficial results were not obtained with the vasoconstrictor solution when it was used alone. The results obtained warrant further study of the use of this drug for common colds.

Tyrothricin nose drops are far from being a specific for head colds; we should be the first to deny any such proposal. However, it is the most successful therapeutic agent which we have used to date, and as such it seems worthy of more widespread trial.

Case Reports

CYSTS OF THE MASTOID BONE

JULES G. WALTNER, M.D., NEW YORK, and SAFA KARATAY, M.D., ISTANBUL, TURKEY

A SMALL NUMBER of cases of cyst involving the mastoid process have been reported in the literature.[1] Microscopic studies in these cases showed a cyst of either a dermoid or a sebaceous character. In some of the cases of dermoid cyst mentioned in the literature bony destruction of the mastoid bone was present, while in others the cyst was located in the subcutaneous layer only. The case of cyst of the mastoid process described in this paper is presented because no report of a similar case was found in the literature and because the clinical course and the examination in no way indicated the extensive pathologic process involving the dura and the petrous bone.

REPORT OF A CASE

A 17 year old white youth noticed a painless swelling behind his left auricle three months before his admission to the Presbyterian Hospital, New York. He had never experienced any pain or discharge of his ears. He did not remember any loss of hearing, even of a transitory character.

Both external auditory canals and drums were normal. There was a fluctuating, slightly tender, moderately raised swelling, measuring 20 mm. in diameter, just posterior to the insertion line of the left auricle. Nose, nasopharynx, pharynx and larynx revealed no pathologic condition. Transillumination showed clear sinuses. Heart and lungs were normal. The Kline test was negative. The serum calcium was 8.7 mg. per hundred cubic centimeters; the serum phosphatase, 3.2 Bodansky units. The audiogram showed normal hearing. Roentgen studies (fig. 1) of the left temporal bone revealed a large bony defect over the mastoid and petrous bones. This bony defect was quite similar to that of an old mastoidectomy. The right mastoid process roentgenologically showed no pathologic change.

On evidence of the clinical examination and of the roentgen studies, the diagnosis of a retroauricular cyst was made.

From the Department of Otolaryngology of the Columbia University College of Physicians and Surgeons and the Presbyterian Hospital.

The authors were assisted by grants from the Research Council of the American Otological Society and the Hayden-Coakley Fund.

1. Johnston, R. H.: A Large Dermoid Tumor of the Mastoid, J. Eye, Ear & Throat Dis. **8**:124, 1903. Levesque, L.: Contribution à l'étude des Kystes dermoides de l'apophyse mastoide et du pavillon de l'oreille, Thesis, Paris, 1907. Aubriot, J.: Sur un kyste congenitale de la region mastoidienne, Compt. rend. Soc. de biol. **85**:517, 1921. Cini, C.: Quiste dermoideo de la región mastoidea, Semana méd. **1**:862, 1930. Comes, S.: Su una cisti della regione mastoidea, Arch. ital. di otol. **45**:559, 1934. Amersbach, K.: Mucocele des Warzenfortsaatzes Epidurales Cholesteatom des Schläfenbeines, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**: 272, 1934. Weaver, D. F.: Unusual Cyst of the Mastoid, Arch. Otolaryng. **37**:854 (June) 1943.

Under local anesthesia a retroauricular incision was made, easily exposing the dome of the bulging cyst under the skin and periosteum. The circumference of the cyst was adherent to the underlying mastoid bone, and the attempt to separate the cyst resulted in its rupture. From 15 to 20 cc. of dark brownish, lipiodol-like fluid under pressure was seen in a large cavity which extended into the depths of the mastoid bone. The cortex of the mastoid bone was missing in an area measuring 30 mm. in diameter just below the level of the mastoid antrum

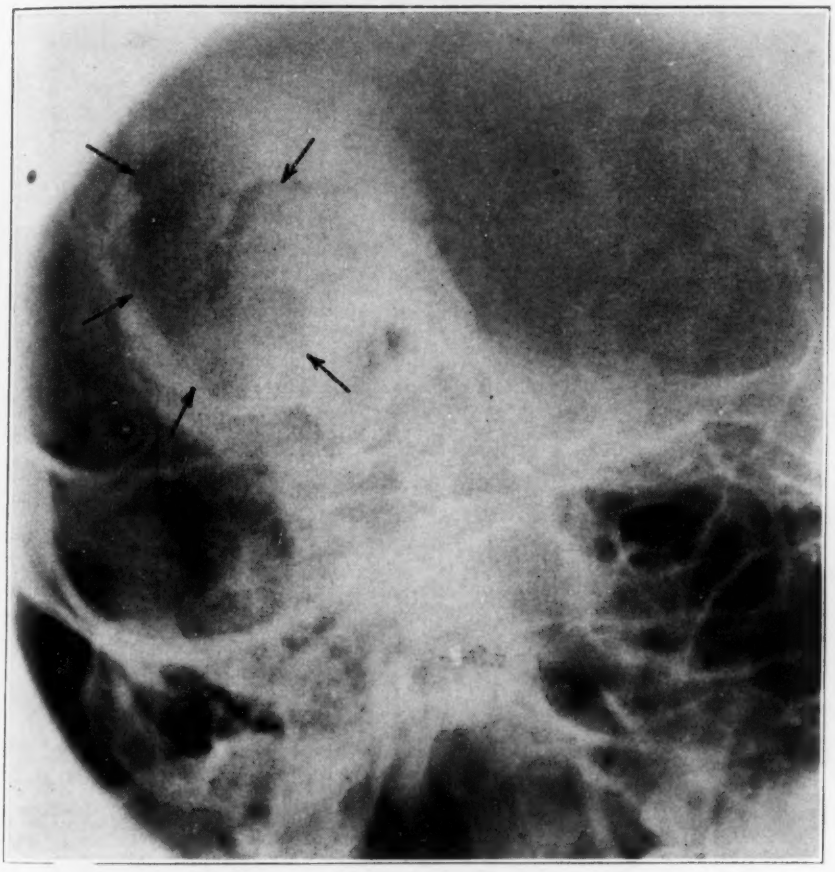

Fig. 1.—Roentgenogram of the left mastoid process (Law's position). Arrows indicate the bony defect.

and 6 to 7 mm. posterior to the external auditory canal. This bony defect led into a deep cavity which proved to be several times larger than was indicated by the size of the cortical defect. Exploration of the entire cavity was postponed pending the result of the histologic studies of the cyst wall.

Histologic examination of the cyst wall revealed a cyst lined by an epithelium similar to that of the middle ear. The lipiodol-like material and cholesterol crystals present most likely represented a product of degeneration of portions of the epithelial lining (fig. 3).

Cultures from the cyst showed no growth.

A second operation was made two days after the first one, with the patient under general anesthesia. After the pathologic bony defect of the cortex had been enlarged, a huge cavity was exposed which occupied (1) the entire mastoid

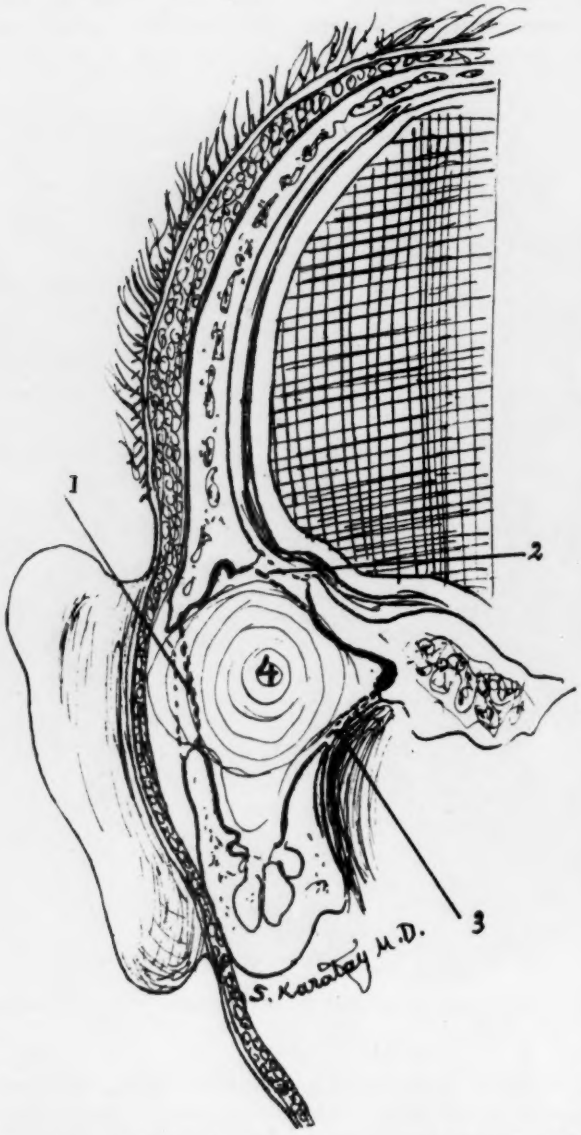

Fig. 2.—Coronary section: (*1*) defect of the mastoid cortex; (*2*) destruction of the inner table of the tegmen antri; (*3*) destruction of the inner table at the sinodural angle; (*4*) the mastoid cyst.

process and deeply indented (2) the petrous portion of the temporal bone and (3) the neighboring area of the occipital bone (fig. 2). The posterior bony wall

of the external canal was partially destroyed. The wall of the cavity was covered by firm granulation tissue, which filled out the mastoid antrum and the enlarged aditus. These granulations were directly continuous through two bony defects in the inner table with a similar tissue extensively covering the dura of the middle and posterior cerebral fossae. One of these pathologic bony defects was located in the tegmen antri, the other in the sinodural angle. The inner table was brittle

Fig. 3.—Wall of cyst: (*a*) cuboidal epithelial lining; (*b*) supporting edematous connective tissue containing numerous lymphocytes and plasma cells.

and was easily removed with rongeurs. After a large exposure of the cerebral and cerebellar dura the granulation tissue could be separated everywhere from the slightly injected dura. The sigmoid sinus was either obliterated or absent in its lower half, for blood could be aspirated only at the level of the sinodural angle.

The postoperative course was uneventful.

Histologic examination of the tissue covering the deep portion of the cystic cavity showed it to be heavily infiltrated by mononuclear leukocytes, most of

which were plasma cells. There were numerous Russell bodies. In many places the epithelium was cuboidal and showed the characteristics of the mucosa of the middle ear. There were large collections of cholesterol crystal clefts, associated with foreign body giant cells and blood pigment. There were several collections of large foamy phagocytes. In some places small clumps of darkly staining epithelial cells were seen surrounded by inflammatory tissue (fig. 4).

The tissue which covered the dura proved to be gramulation tissue containing collections of cholesterol crystal clefts and a moderate number of mononuclear cells. No epithelial elements were found in this gramulation tissue. There were

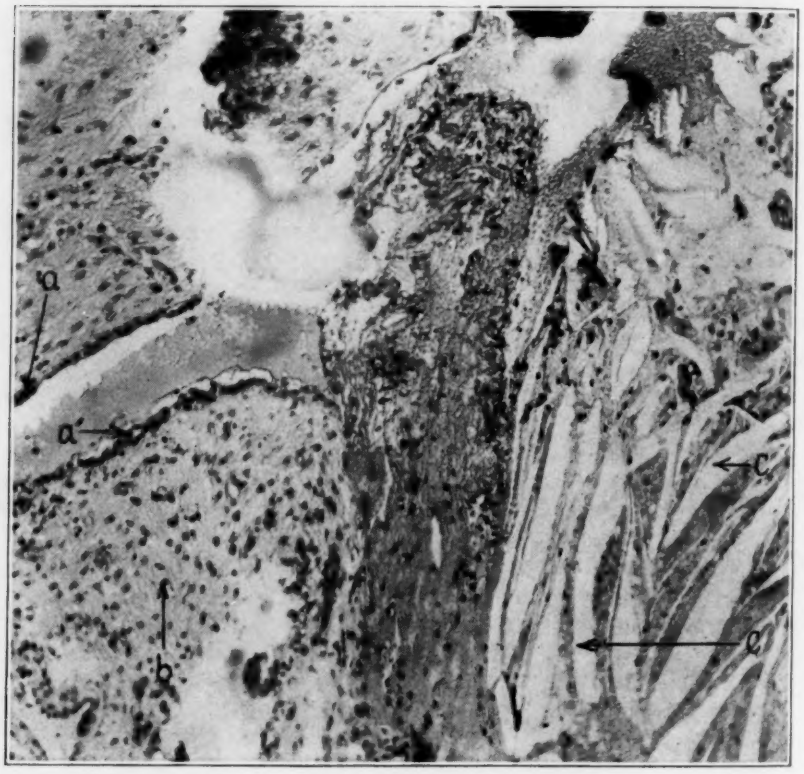

Fig. 4.—Deep portion of the cyst wall covering the enlarged mastoid cavity: (*a*) low cuboidal epithelial lining; (*b*) supporting stroma heavily infiltrated by mononuclear leukocytes; (*c*) cholesterol crystal clefts.

numerous small lamellas of bone, irregular in thickness and surrounded by the described granulomatous tissue (fig. 5).

Sections of the decalcified pieces of the inner table showed no involvement of the bone.

COMMENT

In this peculiar case there is presented a cyst lined by epithelium of the middle ear type which, because of partial degeneration of its epithelial lining, has become surrounded by inflammatory, granuloma-

tous tissue. This inflammatory tissue, probably aided by pressure atrophy, destroyed a good portion of the mastoid and petrous bones.

The pathogenesis of this unusual cyst cannot be established with certainty. A developmental anomaly that arose during the course of pneumatization seems to give the most probable explanation. During the first stage of pneumatization of the mastoid process a budding pneumatic cell lined with epithelium might have been detached from the

Fig. 5.—Granulation tissue covering dura: (*a*) mononuclear cell infiltration; (*b*) cholesterol crystal clefts.

mastoid antrum or the adjoining pneumatic cells. After the separated cell lost its communication with the main air-containing cavities cystic fluid probably accumulated, and pressure was exerted by the cyst on the surrounding bony structures and this caused the bony defect. In the course of the development of the lungs a similar process may result in the formation of an anomaly which is known as congenital cystic lung.

Although the patient's history was noncontributory from the point of view of infections of the middle ear, and clinical examination failed to reveal any evidence of a healed or present infection of the middle ear, an inflammatory process cannot be ruled out as a factor in the development of this cyst.

The progressive symptom-free destruction which so extensively involved the cortex of the mastoid process, the petrous bone and the inner table is noteworthy. It is evident that in a number of years it would have come to a more extensive encroachment on the endocranium.

SUMMARY

A cyst of the mastoid and petrous bones is presented as the first to be recorded in the literature.

Histologic studies clearly separate this cyst from a larger group of dermoid cysts located in this area.

The cyst destroyed most of the mastoid process, formed two large defects in the inner table covering the cerebellum and the temporal lobe, and extended into the occipital bone.

Total removal of the cyst and of the granulation tissue covering the dura was achieved by means of surgical measures.

The pathogenesis of this cyst is explained as a developmental anomaly which occurred during the process of pneumatization. It can be compared to the congenital cystic lung, which represents a disorder of budding occurring during the development of the lung.

850 Park Avenue.

ADENOMA OF THE TRACHEA

MORLEY T. SMITH, M.D., NEW ROCHELLE, N. Y.

THAT adenoma of the trachea is comparatively rare is indicated by the few cases reported in the literature as well as by the personal experiences of several authorities working in large clinics.

REPORT OF A CASE

M. F., a white woman aged 37, was first seen in a medical clinic on Dec. 13, 1940. Her chief complaint at that time was asthmatic attacks, from which she had suffered for about two years. She also complained of frequent infections of the upper respiratory tract with postnasal discharge and occasional frontal headaches. She stated that she had attacks of dyspnea with hacking cough occasionally and that these were inclined to be more severe at night, lasting from a few minutes to several hours. There was no history of hemoptysis or excessive sputum. Her past history was essentially irrelevant.

Physical examination at this time showed no abnormality apart from the upper respiratory tract except obesity, the patient weighing 245 pounds (111.2 Kg.). She was referred to the nose and throat service, where the findings were as follows: She had hyperplastic rhinosinusitis, with polypoid changes, and secondary suppurative infection. At rest the patient breathed without difficulty, but on exertion she showed some signs of dyspnea, with an occasional wheeze. Indirect laryngoscopy showed the larynx to be normal in appearance and motility. In the subglottic region there was a slightly irregular, lobulated tumor mass, covered with normal-appearing mucous membrane. No ulceration was noted. The tumor appeared to be attached by a broad base to the right anterolateral tracheal wall. It was immediately subglottic and occupied about two thirds or more of the tracheal lumen. It did not move on respiration. The regional lymph nodes were not enlarged.

Laboratory Findings.—A roentgen study of the trachea showed a well defined, smooth-appearing mass with a broad base attached to the right anterolateral tracheal wall just below the right cord. This tumor, as shown in figure 1, occupied about two thirds of the tracheal lumen. Roentgen study of the chest revealed no abnormality. The blood and the urine were normal. The Wassermann and Kline tests were negative.

Treatment.—On Jan. 18, 1941, after anesthesia had been induced by infiltration of a 1 per cent solution of procaine hydrochloride, a midline incision was made from the hyoid bone to the suprasternal notch. The pretracheal muscles were separated, and the isthmus of the thyroid gland was separated from the trachea and divided. The first four tracheal rings were divided in the midline, also the arch of the cricoid cartilage. The latter procedure was necessary in order to expose properly the attachment of the tumor. The tumor was well defined and attached by a broad base immediately beneath the right vocal cord. The tumor was removed with a nasal snare in one piece, and the base was cauterized with the coagulating current. There was little bleeding during this procedure, and the tumor did not appear invasive. The wound was closed in layers, and the postoperative course was entirely uneventful.

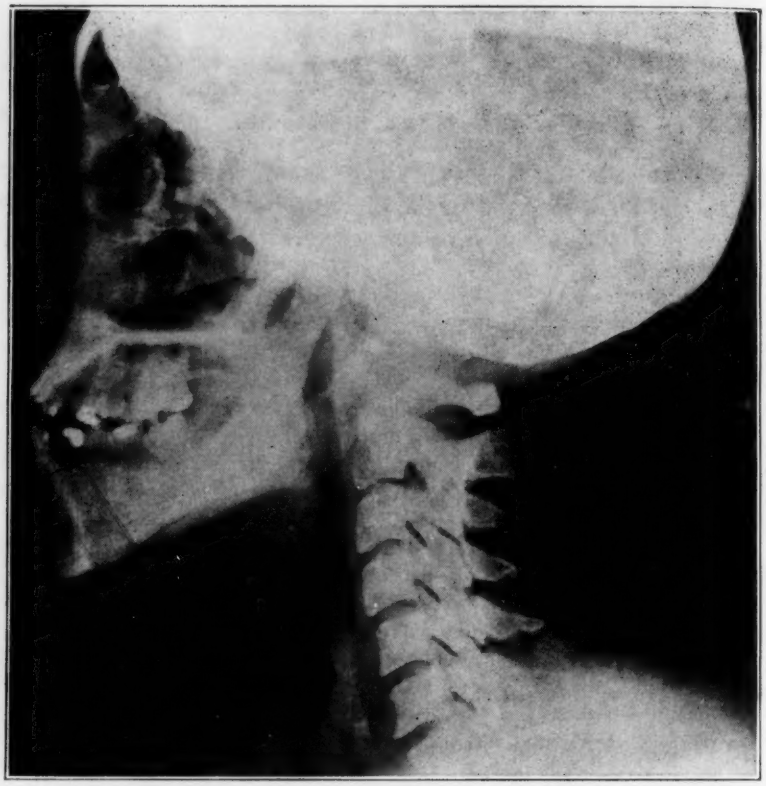

Fig. 1.—Roentgenogram showing well defined, smooth-appearing mass, with broad base, attached just below the right cord to the anterolateral tracheal wall.

Fig. 2.—Section of the tracheal tumor.

The dyspnea disappeared after the removal of the tumor, and there has been no recurrence.

The patient has been followed regularly in the tumor clinic. She was last seen in February 1946, at which time there were no signs of recurrence, and the patient was asymptomatic.

Pathologic Report.—"The specimen consists of a raspberry-shaped, irregularly rounded mass measuring about 1.5 by 1 by 0.5 cm. Its surface is smooth and glistening except at the site of removal. On section a solid, yellow-white somewhat elastic surface is seen."

Histologic Observations.—"Underlying the respiratory epithelium there is a well circumscribed adenomatous nodule. The growth appears to be orderly, composed of uniformly small basophilic cells, which are arranged in small alveoli, and these frequently are confluent and coalescent. Stroma is relatively scant and is hyaline in type.

"Diagnosis: The growth is a benign adenoma of bronchial type."

This specimen was also examined by five other experienced pathologists, and their reports were as follows: (*a*) benign bronchial adenoma, likely to recur; (*b*) adenocarcinoma; (*c*) adenocarcinoma arising from minor salivary glands; (*d*) benign mixed tumor; (*e*) aberrant tumor of the thyroid gland, with no evidence of malignancy.

SUMMARY

A case of adenoma of the trachea is presented, with successful removal by the external surgical approach. Five years have elapsed since removal of the tumor and there is no evidence of recurrence or metastasis at this time (February 1946). In view of the time elapsed since the operation, it would seem that this tumor might be classified as a benign tracheal adenoma.

421 Huguenot Street.

Abstracts from Current Literature

Ear

A Group Pure Tone Hearing Test. Scott N. Reger and Hayes A. Newby, J. Speech Disorders **12**:61 (March) 1947.

Forty ear phones were connected to the output of a pure tone audiometer, the oscillator circuit of which was suppressed or released according to a predetermined sound stimulus pattern by a technically operated tone interrupter while an attention-focusing light was turned on. The mechanism of the attention-focusing illumination consisted of five bakelite disks, 4 inches (10 cm.) in diameter and ¼ inch (6 mm.) thick. The disks were rotated at the rate of six revolutions per minute, and were cut with one to four projecting teeth on their perimeters, which actuated microswitches. The disk associated with the 60 watt attention light illuminated the bulb five-tenths second before the first tone of the sound pattern was produced. While the light was off, the operator turned a selector switch to select any one of the desired frequency and intensity levels. The operator followed a key, which showed in the proper intensity level settings the number of interruptions for every square of the text blank. The top square of each column represented a tone of 40 decibels above the normal. Proceeding down each column, the tone was presented successively 40, 30, 20, 15, 10 and 5 decibels above normal. The frequencies tested were 256, 1025, 2048 and 4096 cycles per second. Preliminary experimentation was undertaken with a group pure tone test used in conjunction with 10 "permoflux" dynamic ear phones, produced by the Permoflux Corporation, Chicago, instead of the 40 Western Electric Company phones. With the "permoflux" phones it was possible to test the acuity through 8192 cycles. Thirty additional "permoflux" phones will be obtained in the future. On the basis of this group test all entering students of the State University of Iowa with serious losses of any of the four frequencies were screened out for thorough examination. Approximately 2,400 pupils in the Iowa City public school system will be tested this year.

Palmer, Wichita, Kan.

Chronic Mastoiditis with Cholesteatoma and Stenosis of the External Auditory Meatus. H. I. Lillie and James B. McBean, Minnesota Med. **30**:161 (Feb.) 1947.

Lillie and McBean state that stenosis of the external auditory canal presents a diagnostic problem in cases in which pain or discharge is present. They report 2 cases to direct attention to this problem and to emphasize the importance of adequate drainage in the treatment of chronic otitis media.

Stenosis of the canal may be congenital, in which case it is an asymptomatic condition. Acute otitis externa is a disease of short duration, with the pain localized in the ear itself and with pain on movement of the auricle or pressure of the tragus. Hearing is found to be normal. However, when stenosis is produced by chronic otitis media there is a long history of discharge of the ear. The pain is more deep seated. The patient complains of a deep-seated headache, and the hearing is much diminished. There is no pain on the movement of the auricle, but deep tenderness of the mastoid process may be present.

In the first of the 2 cases reported, operation showed much pus in the mastoid process proper and an extradural abscess. There was also a large cholesteatoma. Adequate removal of all infected bone was accomplished, and a radical mastoidectomy was performed, with large skin flap being made.

In the second case, also, the stenosis was severe. Roentgen examination revealed partial sclerosis of the mastoid process. Operation revealed a destructive process which had advanced as far as the dura of the middle fossa in two places. The middle ear was packed with cholesteatoma. A radical mastoidectomy was performed, and there was perfect and uneventful recovery.

The authors feel that in both of these cases the pathologic conditions that existed in the mastoid bone and next to the dura were aggravated by the lack of adequate drainage. They feel further that in such cases surgical intervention is urgent, in order to prevent intracranial extension of the infection. LEWY, Chicago.

Pharynx

TONSILLITIS AND PHARYNGITIS. COMMISSION ON ACUTE RESPIRATORY DISEASES, J. A. M. A. **133**:588 (March 1) 1947.

The Commission on Acute Respiratory Diseases has undertaken a study of exudative tonsillitis and pharyngitis of unknown cause observed at the Regional Hospital, Fort Bragg, N. C., during the spring of 1943, the spring of 1944 and the winter of 1945.

In this an attempt was made to use precise methods to demonstrate the etiologic factor in any given case, since the assumption has been commonly made that the exudative type of tonsillitis and pharyngitis is caused by beta hemolytic streptococci.

Clinical, bacteriologic and serologic studies were integrated. There are several charts and case reports.

Some cases showed a more severe clinical course, and these were found to be due to streptococci; other cases showed almost similar lesions in the fauces and the pharynx, but were definitely milder in course, and were proved to be non-streptococcic in origin. In at least one of the charts it was noted that the correct diagnosis was entirely dependent on the laboratory tests.

Emphasis has been laid on the observation that the results of bacteriologic tests alone may not always be adequate for diagnosis. Serologic tests should also be carried out, for many subjects, both normal ones and those suffering with respiratory disease, harbor hemolytic streptococci in their throats but do not exhibit antibodies for streptococci. The commission summarizes its report as follows: "Exudative tonsillitis and pharyngitis of unknown origin should be differentiated from streptococcic infection of the throat. The clinical differentiation can be made with reasonable accuracy in many cases but not in all. Accurate diagnosis demands the use of cultural and serologic methods. The salient features of these two types of exudative tonsillitis and pharyngitis have been presented, and cases have been reported to illustrate the variations in appearance and the difficulties in diagnosis."

GORDON, Philadelphia.

EFFECT OF SMOKING ON THE CALIBER OF THE SUPERFICIAL BLOOD VESSELS IN THE UVULA AND THE SOFT PALATE. P. H. HOLINGER, R. G. RIGBY, A. H. ANDREWS JR., L. H. JAMES and C. W. LENTH, Laryngoscope **56**:762 (Dec.) 1946.

An effort is made to determine accurately the effect of cigaret smoke on the blood vessels of the uvula and the soft palate. In previous work of this sort the operator's judgment was a factor; here it is not.

In addition to five leading brands of cigarets, three specially prepared types of cigarets were used: (1) glycerin-treated, (2) diethylene glycol–treated and (3) containing no humectant.

A special camera and special equipment were used, which permitted the making of fairly exact measurements of the vessels as projected on a screen.

Much information was gathered, but on analysis it proved not to be statistically significant. One of the reasons why the results are inconclusive is that the systemic effect of nicotine is the opposite of its local effect.

The authors conclude that the local irritation caused by smoking cigarets cannot be evaluated by measuring the diameter of blood vessels or by observing the attendant gross color changes.

HITSCHLER, Philadelphia.

Larynx

THE TREATMENT OF CARCINOMA OF THE LARYNX. H. F. HARE and W. B. HOOVER, Eye, Ear, Nose & Throat Monthly **26**:32 (Jan.) 1947.

A plea is made for early diagnosis and treatment of laryngeal carcinoma. Since hoarseness is the only early symptom of intrinsic laryngeal cancer, any hoarseness persisting for more than three or four weeks must be investigated by a trained laryngologist. Early differential diagnosis can be made by laryngeal biopsy, serologic study and roentgen examination of chest and larynx.

Laryngofissure is the treatment of choice for early stages. Radiation will cure a reasonable percentage of lesions that are too far advanced for laryngofissure; laryngectomy can still be carried out if radiation therapy fails. During and after the course of radiation treatment the laryngologist should make careful examinations of the larynx. When the lesion does not disappear either by the end of treatment or within two months, laryngectomy is advised.

The authors describe the radiation treatment which they have been using since 1939 and present tables describing the results obtained in their cases for the two five year periods before and after that date.

To avoid failures of radiation therapy it is necessary to insure that a uniform field of irradiation is obtained and that the cone is well centered. Methods of achieving these aims are described in detail along with the technic and the dosage.

JENNES, Waterbury, Conn.

LARYNGECTOMY AND ITS PSYCHOLOGIC IMPLICATIONS. JAMES SONNETT GREENE, New York State M. J. **47**:53 (Jan. 1) 1947.

Billroth is commonly credited with having performed the first successful laryngectomy in 1870. Many surgeons who followed his lead were beset by failures. Greene divides the experience into an initial phase which had to do with an effort to improve surgical technic, a second phase wherein surgical treatment was supplemented by an effort to restore some kind of voice through mechanical appliances, and a third phase which had to do with rehabilitation through teaching and psychic factors. There is a terrific psychic trauma in every person who faces laryngectomy. Greene asks his patients five questions: (1) fear or anxiety felt during the period of hoarseness, (2) the "reaction" when loss of the larynx proved inevitable, (3) the emotional "upset" on entering the hospital for operation, (4) the "feeling" when recuperating and finding no response on trying to talk and (5) the effort to resume old associations. In Greene's clinic the patients meet others who have also undergone laryngectomy, and this reduces the psychic shock. Some 200 patients have been studied, 70 of them in detailed fashion. Certain recommendations as to management are set down: 1. The patient must be told fully and frankly just what he is to expect, and thus be enabled to withstand his misfortune. 2. He must be introduced to a "recovered patient" who has developed a useful voice. 3. This same person should call on the patient soon

after operation to reassure him, 4. The surgeon should recommend voice training as soon as feasible. 5. Class teaching of laryngectomized persons is a valuable factor, along with personalized training.

VOORHEES, New York.

Nose

RECONSTRUCTION OF THE NASAL SEPTUM. W. H. STEFFENSEN, Plastic & Reconstruct. Surg. **2**:66 (Jan.) 1947.

Steffensen has performed reconstruction of the nasal septum in a series of 311 cases. The results have been uniformly good. A warning is given against the injudicious use of submucous reaction. He finds that it is not necessary to remove the septal cartilage completely. A procedure is described for combining the septum correction and the reshaping of the nasal framework, when the full length of the septum is not involved. The author states that reconstruction of the septum can be done on any type of septal deformity and at any age. He does not agree that it is necessary to wait for the full growth of the nose. The greater part of this report follows methods already reported by others.

SELTZER, Philadelphia.

RECONSTRUCTION OF THE COLUMELLA. S. SALINGER, Plastic & Reconstruc. Surg. **2**:87 (Jan.) 1947.

The author's technic of surgical treatment for total loss of the columella requires three or four operations. The flap for the graft is taken from the acromiopectoral region and moved either to the angle of the jaw or directly to the field of operation. This method avoids scarring the face, and the scar at the angle of the jaw is not noticeable. He describes a shorter method in which the tube flap is taken from the neck parallel to the sternocleidomastoid muscle. He finds that it is better to implant the tube at the base rather than at the tip of the columella, as the circulation is better in that area, and the lack of traction obviates the risk of depressing the nasal tip. The use of a cartilage implant may or may not be necessary.

SELTZER, Philadelphia.

ANOTHER EXTERNAL NASAL SPLINT. J. G. HEBERT, Plastic & Reconstruct. Surg. **2**:159 (Jan.) 1947.

Hebert's nasal splint is made up of three parts: (1) a plaster of paris bandage forming an around the head cast; (2) a light plaster mold which covers the dorsum of the nose closely and extends downward to include the nasolabial angles, to minimize postoperative swelling; (3) a spring action device which has a universal joint making it independent of forehead-scalp movement. Counterpressure is derived not from the forehead but from the occiput. The splint was originally designed with the use of a plaster bandage, the spring of a large safety pin and suitable parts of a mouse trap.

SELTZER, Philadelphia.

Miscellaneous

VIRAL INFECTIONS OF THE RESPIRATORY TRACT. HOBART A. REIMANN, J. A. M. A. **132**:487 (Nov. 2) 1946.

Reimann discusses acute, self-limiting, chiefly air-borne diseases of the respiratory tract which are known or presumed to be of viral origin, usually called cold, grip and influenza.

He discusses in moderate detail the epidemiology thereof, emphasizing that exhaled particles floating in the air disseminate the viruses, and comments on the

general lack of immunologic understanding of these types of infections, excepting influenza A and B.

There are clarifying comments on the terminology of the various respiratory infections.

In a critical appraisal of the current treatment he stresses the futility of the many measures used, both general and local. As regards preventive measures, he frowns on vitamin therapy, sun tanning, ultraviolet ray therapy, face masks, the so-called hardening process, cold vaccines and tonsillectomy. Concerning the last, he states: "Obstructive adenoids, however, did appear to bear a relationship to frequent colds." He then discusses rational general and local therapy in the management of the varying symptoms that arise in the respiratory system, as well as rational preventive measures.

This abstracter has always had a high regard for Reimann, a distinguished internist, and though many otolaryngologists may not agree with the author in all that he says, nonetheless any one carefully reading the original article will be amply repaid.

GORDON, Philadelphia.

INFECTIOUS DISEASE. HOBART A. REIMANN, J. A. M. A. **132**:969 (Dec. 21) 1946.

This distinguished internist reviews the valuable contributions of antibiotic agents as used in the control of respiratory infections as well as the intercurrent infections that so often arise during the period of incidence of degenerative diseases.

He deplores the indiscriminate use of penicillin. Much of this valuable antibiotic is wasted, since it is given for diseases on which it is known to have no effect; fever is often the only indication for its use. Often the doses are too small or the treatment too interrupted, or they are too large or are given longer than is necessary; oral therapy requires two to four times the amount needed for parenteral therapy, or it may be used as a prophylactic too often in the absence of evidence of its usefulness for this purpose. Though these abuses are not serious from the point of view of the patient, except for an occasional untoward effect, they represent economic waste and are in part responsible for recent shortages. Furthermore, there is the likelihood that, with widespread indiscriminate use, penicillin-resistant bacteria will arise, so that after several years the latter will cancel the curative effect of penicillin.

Reimann gives several absolute indications for the use of penicillin.

Streptomycin has but limited use. It is of value in the treatment of tularemia, some infections caused by Pfeiffer's bacilli and some types of urinary infections. Other studies of this antibiotic have not been sufficiently advanced to enable one to draw conclusions.

GORDON, Philadelphia.

THE LABILITY OF BONE. Editorial, J. A. M. A. **133**:110 (Jan. 11) 1947.

The physical characteristics of bone are more obvious than its metabolic activity. The fact that bone is hard and rigid and appears so admirably to fulfil its mechanical functions is likely to turn attention from its capacity as a labile reservoir of mineral salts. Long before actual experimental demonstration the pathologist was aware of such abnormalities of mineralization or of decalcification as rickets, scurvy, osteitis fibrosa cystica and osteoporosis. More than a decade ago it was demonstrated that restricting experimental animals to a salt-poor ration not only reduced the proportion of total bone ash but actually altered the chemical composition of the salt deposited in the growing skeleton. The process whereby minerals are withdrawn from the skeleton is influenced by certain of the hormones. The thyroid hormone has been used to remove lead from the body after lead poisoning; the heavy metal appears to be mobilized from the bone along with the calcium phosphate with which it was deposited. Again, the hormone of the parathyroid glands has a definite effect on bone calcium; deficiency of this substance results in hypocalcemia and resultant tetany, whereas overproduction of the

hormone produces hypercalcemia and net losses of calcium and phosphorus, salts which come from the skeleton.

The estrogenic hormones cause bone salts to be shifted from the symphysis pubis to the long bones in certain species, with the result that the breaking strength of these leg bones is greatly increased and the chemical composition of the bone salts is altered. Purely physiologic evidence of the intense activity of the femur that results from the administration of estrogens has recently been reported; the roentgen diffractograms of sections of these bones show complete disorientation of bone salt crystals in the cortex. The same method of study has indicated that the faulty mineralization of the epiphysis in rickets is characterized by disorientation of the bone salt crystals and that this abnormal structure persists after clinical healing is complete. By means of a number of different chemical and physical experimental approaches it has been demonstrated that the skeleton is surprisingly labile; bone salts can be withdrawn from this reservoir under a variety of influences, as well as deposited when circumstances are favorable. This fundamental concept has helped to explain certain clinical conditions and should be kept in mind in the therapeutic and nutritional approach to medical problems.

GORDON, Philadelphia.

FOCAL INFECTION. Current Comment, J. A. M. A. **133**:111 (Jan. 11) 1947.

In his presidential address to the Institute of Medicine of Chicago Dr. Ernest E. Irons discussed focal infection from the time when an Assyrian physician-priest in the seventh century told his king that the constitutional ailment from which he suffered was related to infected teeth to the revival of this doctrine by Frank Billings in the period from 1912 through 1916.

Earlier investigators had arrived by empiric methods at the concept of a relationship between infection of the teeth and disturbances occurring elsewhere in the body. With the coming of the modern era of bacteriology the infecting organism could be traced from its focus in the teeth or the throat or elsewhere in the body to tissues and organs far removed. Focal infection was no longer a theory but an established process. The difficulty of determining the exact nature of the relation which obtains between a focus of infection and chronic arthritis is obviously far greater than that of tracing such an infection from a tooth to the heart or to the kidney. Today the desirability of finding and removing local and often symptomless infection is generally recognized. However, removing great numbers of teeth and operating on the nose and the throat on the chance that there is a relation between infection and chronic arthritis is no longer considered a scientific procedure. True, removal of chronic infections, such as alveolar and peritonsillar abscesses, may results in permanent cessation of arthritic attacks, but new technics of dental care and the development of specific chemotherapy and antibiotic therapy make available other approaches to this problem. In retrospect the intensive study carried on by Dr. Frank Billings and his associates, among them conspicuously Dr. Ernest E. Irons, produced not only observations of great importance in modern diagnosis and therapy but also concepts and procedures of significance in the control of other diseases.

Among the chief of these by-products is the more careful examination of patients. Today the concept of focal infection has been integrated into the practice of medicine. One speaks no longer of the theory of focal infection; one recognizes focal infection as a definite pathologic condition requiring scientific diagnosis and treatment.

GORDON, Philadelphia.

ALLERGIC PAROTITIS. GEORGE L. WALDBOTT and J. J. SHEA, J. Allergy **18**:51 (Jan.) 1947.

Three patients with bronchial asthma exhibited recurrent swelling of the parotid glands. Asthma preceded the swelling in 2 of the patients, while in 1 the recurring parotitis had been present for some years prior to the onset of the

bronchial asthma. The allergic background of both the asthma and the parotid swelling is not definitely known. Allergic edema, and actual mucous edema of the glandular tissue, obstructing the parotid duct, are the two possibilities. The authors raise the question as to whether nonspecific recurrent parotid swelling, often described as infectious parotitis, may not be of allergic origin with superimposed infection.

FRIEDBERG, Chicago.

INTENSIVE GROUP THERAPY IN SPEECH REHABILITATION. OLLIE L. BACKUS and HARRIET M. DUNN, J. Speech Disorders **12**:39 (March) 1947.

This is a report on sixteen intensive programs with 198 cases of different speech disorders. Programs were carried out in a six week session, from 9 a. m. to 4 p. m. The intensive group therapy is radically different in scope and plan from traditional clinical practice. It necessitates a different administrative setup, employs different teaching materials and requires special training on the part of clinicians. Intensive group therapy provides good opportunities to study the patient and his particular needs, to convince him of his problems and needs and to teach him how to carry over new speech patterns into real life situations. Intensive group therapy has advantages as an instrument of teacher training and is a useful instrument for research in the clinical practice of speech rehabilitation. Persons with various kinds of defective speech have in common problems relating to speech breakdown and relapse. Speech rehabilitation for all patients is a process of gaining voluntary controls. Certain basic technics should be applied in the treatment of all speech disorders. Learning proceeds faster if the concept of forming rich associations in learning is considered to mean employing a few that are strong rather than employing many. Numerous illustrative cases are presented.

PALMER, Wichita, Kan.

ORAL PENICILLIN IN OTOLARYNGOLOGY. LIERLE and PAUL, Laryngoscope **56**:352 (July) 1946.

Lierle and Paul review the results of the oral administration of penicillin in diseases of the ears, nose and throat.

One hundred and twenty-four cases were studied. These included most of the usual diseases encountered by the otolaryngologist. As the authors point out, the numbers of cases are so small, and the diseases discussed are of such a nature, that no definite conclusions can be drawn. In most cases, doses given on an empty stomach, two to three times the hypodermic dose, seemed to produce satisfactory results. Chronic conditions did not respond as well as acute conditions. Few toxic reactions occurred, and these were of a mild nature.

HITSCHLER, Philadelphia.

PREVENTIVE MEDICINE IN THE CANCER FIELD. C. P. RHOADS, New York Med. **3**:19 (April 5) 1947.

Rhoads states that the study of cancer may well be the prototype for research in any other field of medicine. The periodic physical examination is extremely important in finding cancer, and many other diseases. In the Strang Cancer Prevention Clinic at Memorial Hospital for the Treatment of Cancer and Allied Diseases, 140 cancers were found in 10,000 persons, and other conditions requiring medical care also were seen. Of persons declared to have been in good health, 26 had early cancer. The cost of such determinations is between $7.50 and $10, but the check-up is exceedingly thorough. As to "cancerophobes," it seems better to make the public cancer conscious, regardless of "fear" as a fixed entity. The

corollary is that if every persistent hoarseness were investigated by the laryngologist, fewer patients would come to complete laryngectomy too late, and many early cancers would be discovered while still amenable to partial resection or irradiation.

As to the cost of regular, periodic cancer-preventive work, "It is my belief that $10,000 spent to examine 1,000 well women . . . is money well spent. Few patients are cared for at a (public) cost of less than $1,000 each, and certainly in half of them the money is thrown away, in heroic but futile activity." This article deserves a wide reading, and it will serve to make the doctor, as well as the patient, cancer conscious, which is certainly a desideratum.

VOORHEES, New York.

BILATERAL CLEFT LIPS. L. W. SCHULTZ, Plastic & Reconst. Surg. **1**:338 (Nov.) 1946.

Schultz presents a method for closure of congenital bilateral cleft of the upper lip. It is emphasized that the intermaxillary bone should be placed in normal position at about the age of 1 month. When done according to the author's method of removing both mucous membrane and periosteum and fixing the parts bone to bone, the results have been satisfactory. No. 4-0 Nylon is passed around the intermaxillary segment, and the parts are joined by means of mattress sutures of no. 6-0 Nylon. This secures bony union. After a one month interval, the soft parts are corrected. The details of this procedure are given and well illustrated.

SELTZER, Philadelphia.

THE USE OF NEIGHBORING TISSUES IN THE CORRECTION OF AN EXTENISVE FACIAL DEFORMITY. W. M. ADAMS, Plastic & Reconstruc. Surg. **2**:105 (Jan.) 1947.

Adams favors using skin adjoining the defect for repair whenever possible—otherwise, from a corresponding area on the opposite side of the face—to insure a similar type of skin. If the wounds are large, several operative steps should be done, not timed too close together. This is designed to allow for shrinking of the skin, the size of the area and the distance the graft must be shifted. The time between operations can be shortened by massage. Very fine subcuticular sutures made with sharp, fine cutting needles are used to prevent stitch scars. After the sutures are removed, the incision is supported by some form of strapping until the tension of the skin lessens.

SELTZER, Philadelphia.

CLEFT LIP REPAIR AFTER AXHAUSEN. H. MAY, Plastic & Reconstruct. Surg. **2**: 139 (Jan.) 1947.

The method, given in detail, is applicable to all types of clefts and to the repair of secondary deformities following primary repair. It assures a firm closure of the cleft and correction of surrounding deformities, including nasal deviation, flattening of the nostril and shortness of the lip, by providing a floor for the nostril, as well as correction of septal and alar displacement and closure of the lip.

SELTZER, Philadelphia.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Francis Lederer, M.D., *President*

Walter H. Theobold, *Secretary*

Regular Monthly Meeting, Nov. 4, 1946

SYMPOSIUM—THE PHYSIOLOGY OF SPEECH

Its Phonetic Aspect. Presented by H. Koepp-Baker, Ph.D.

Its Laryngeal Aspect. Presented by Harold Westlake, Ph.D., Northwestern University.

Teachers of speech have been interested in the anatomy and the physiology of normal breathing as well as in the modifications of breathing which take place during the act of speech. Symptoms of certain disorders of speech are described in terms of irregularities of breathing. In many cases training of speech involves the establishing of breathing patterns which are favorable to good speech.

Presuming to teach proper breathing posed these questions. The factual bases for answers to these questions were ascertained through research.

What changes take place in the thorax during breathing? The most discerning research pertinent to this point was done by Dr. Harlan Bloomer, working under the direction of Dr. Carlton Peirce in the Department of Roentgenology at the University of Michigan. The critical facts are these:

1. The essential movement for inhalation is the upward thrust of the thorax.
2. The twelve ribs move upward together in inhalation and downward together in exhalation.
3. The first two ribs are active even in quiet respiration.
4. The vertical displacement is greatest for the upper ribs and successively less for the lower ribs in order.
5. The latter condition emphasizes the importance of the scalenes as muscles of inhalation.
6. The upward excursion of the twelfth rib in inhalation and its downward excursion in exhalation classifies quadratus luborum as a muscle of exhalation rather than a muscle of inhalation.
7. The angle of the sternum does not change during breathing. The movement is upward and forward during inhalation, and downward and backward during exhalation.
8. The active role of the diaphragm in breathing appears to have been exaggerated. By volumetric changes, diaphragmatic shift may account for 42 per cent of the total exchange of air in the average person. Volumetric changes computed from changes in the configuration of the diaphragm cannot be entirely attributed to the specific activity of that muscle, since widening and upward movement of the circle of origin has its effect on the configuration.
9. The differences between forced and quiet breathing appear to be quantitative rather than qualitative.

How does breathing for speech vary from normal breathing?

1. Varying lengths of phrases make breathing during speech less regular than normal breathing.

2. The relative durations of inhalation and exhalation are reversed. In normal breathing the inhalation phase is relatively long and the exhalation short. During speech the inhalation is short and the phrases of speech are uttered during a much prolonged exhalation.

3. Although there is a tendency for breathing to be more rapid during speech, the average number of breaths taken by a person during continuous speech tends to follow his normal breathing rate.

4. There is no factual basis for believing that breathing during speech varies from ordinary breathing.

In considering the valving at the level of the larynx during the act of speech, two types of exploration have been especially productive: roentgenographic stills and roentgenographic moving pictures showing the changes in the adjustments of the cartilages as well as in those of the soft tissues, and colored moving pictures by which the same adjustments could be observed from the superior view. The questions posed by the teachers of speech as well as the factual bases for the answers are as follows:

Are there definite postures of, or relationships between, the laryngeal structures during the production of specific sounds?

1. Of all the structures involved in the production of speech, the tongue shows the greatest tendency to assume particular postures for specific sounds. The tissues of the larynx show little consistency in their relationships for particular sounds.

Is there a manner of laryngeal valving which will result in desirable vocal tone?

1. Teachers of speech have described two types of vocal attack: the aspirate attack and the glottal attack. In the aspirate attack the true vocal folds approach the midline evenly; sometimes there is a light contact of the vocal processes, but usually they are only approximated. The exhalation appears to begin slightly before phonation begins. There is little contraction of the supraglottic tissues. In the glottal attack the vocal processes come together sharply and there is a marked tendency for the supraglottic tissues (the cushion of the epiglottis and the ventricular bands) to close in. The aspirate attack results in a desirable vocal quality, and the glottal attack, in a sharp, strained quality.

Can vocal misuse of the larynx cause laryngeal irritation?

1. A study in which colored movie photography was used indicated that persons speaking with harsh voices had notable reddening of the vocal lips after a period of continuous oral reading. The same persons, using softer, but equally loud voices, had little or no reddening of the vocal lips during equal periods of oral reading.

2. A second study indicated that ulcers occurring near the vocal processes of the arytenoid cartilages improved after the patients learned to use the aspirate attack and the softer voice described. The subjects used in the experiment all had histories of vocal cords abused during speech.

Its Auditory Aspect. Presented by Raymond Carhart, Ph.D., Northwestern University.

In terms of its social importance, the act of speech is not completed until acoustic stimuli are heard and understood.

The evolution of speech has depended as much on the phylogenic development of auditory capacity as it has on increase of phonatory and articulatory capacities.

Similarly, one does not find in the middle ear or in the cochlea unique developments whose presence in man accounts for his singular position as a talking animal. By contrast, one finds recent structural expansions in the cerebral hemispheres (particularly in the temporal lobes, Broca's area [area parolfactoria] and the association areas) whose evolutionary developments account for human speech. As our immediate phylogenic predecessors underwent cerebral development, their capacity to utilize auditory stimuli for symbolic purposes expanded, and, in conjunction, they increasingly employed as symbols the sounds producible by the vocal mechanism. The end result was speech and language.

Any basic consideration of the auditory aspects of speech must recognize that the ability to interpret sounds as symbols for objects, qualities, actions, relations and ideas is the keystone of human language. The present discussion is concerned with three phases of the total interpretative process: (1) the acoustic features which are the basis for differentiating the auditory patterns of speech, (2) the minimal requirements for auditory interpretation and (3) the variables which modify these minimal patterns.

In terms of their acoustic structure, speech sounds divide themselves into six general classes. The vowel sounds and l, r, m, n and ng possess stable bands of resonance. Differentiation is based on the portions of the frequency range at which these bands occur. Second, diphthongs and blends are characterized by a sweep from one resonance band to another. Third, frictional sounds (like f, s and sh) have broad and fluctuating concentrations of sound. Fourth, voiced frictional sounds (like v and z) combine resonance bands with fluctuating concentrations of sound. Fifth, voiceless plosives (like p and t) possess a short silent period followed by a sharp pulse of sound with wide frequency distribution and a fluctuating character. Sixth, voiced plosives (like b and d) combine the features of voiceless plosives with a low frequency component contributed by phonation.

A fact which has been recognized increasingly in recent years is that a relatively limited range of frequencies suffices for understanding of connected speech—provided the person already has an understanding of the language being spoken. The essential frequency band for this type of hearing is from 500 to 2000 cycles per second. Loss for connected speech generally equals the average pure tone loss (in the ear under test) for the band of 512 to 2048 cycles per second.

The listener can ordinarily "make sense" of connected material without having to perceive all the individual elements of speech or phonemes. Listening situations exist, however, in which precise reception of the entire phonetic pattern is essential to adequate understanding. Therefore, a basic aspect of hearing for speech is phonemic discrimination. Listeners, notably those with auditory impairment, differ markedly in ability to make phonemic discriminations. This ability may be measured with special tests such as the Harvard University Pb-50 lists. Deficiencies of phonemic discrimination arise from three general causes: (1) unfavorable pattern of loss (usually sharp dips in acuity for high frequencies), (2) central deficiencies in interpretative capacity and (3) failure to learn in childhood the interpretation of auditory stimuli.

In general conclusion, the auditory aspects of speech are sufficiently complex so that he who would explore a patient's ability to hear speech must proceed with insight and care. Speech tests are the best means of exploring the patient's ability to interpret spoken language. Furthermore, these tests must be specifically selected to explore particular aspects of the comprehension of speech.

DISCUSSION

DR. SHERMAN SHAPIRO: I should like to have Dr. Westlake tell a little more about the recognition of disorders of speech, and also I should like an opinion from Dr. Carhart as to the scientific value of the word lists compiled by Fry and Kerridge for use in the University of London Clinics, as published in the British medical journals.

DR. E. M. MIKKELSEN: The thought occurred to me that a more simple mechanism than the elaborate anatomic structure described could also deliver excellent results. Professional ventriloquists can dialogue several characters with complete change of voice for each, with perfect enunciation. I have watched Bergen, for instance, from scientific curiosity, and I believe his effort to be entirely glottal.

There have been several instances of dogs trained to say a few primitive words. One which I heard many years ago had been trained in Germany, and pronounced German words, but had to be prompted very much to respond, although anxious to please. Many animals below the status of human beings could probably learn to speak in a simple manner if they had a sufficiently high degree of cerebral development.

DR. JEROME STRAUSS: Much that has been brought out is more scientific than clinical. Yet it bears on something that is important to all. I think Dr. Holinger will bear me out that laryngologists have encountered cases of laryngeal disease or lesion, mainly of hypertrophic type, in which by modification of the use of the larynx and without surgical treatment the lesion disappeared. I have learned to differentiate certain of these cases, but not from the standpoint of the doctor of philosophy; my differentiation is purely clinical. I have patients with acquired malfunction of the larynx, many of whom I have classified to myself as "neckers"; that is, one can tell that they talk from the collar rather than from behind the teeth. I should like to know if there is some way in which the "neck talkers" can be modified.

DR. PAUL HOLINGER: This has been a most instructive and interesting evening. I was about to start my discussion by saying that this was the first otolaryngologic meeting I have attended in five years that did not end with a discussion of the pros and cons of fenestration; but finally Dr. Carhart brought that subject in anyway. Nevertheless, the importance of the work presented this evening has not always been understood or appreciated in the strictly laryngologic medical sense. It has always been a borderline field, sometimes associated in one's mind with a pseudoscientific atmosphere that left one a little reluctant to follow. However, the work presented this evening makes one realize the firm basis which this investigation has, and the extremely thorough nature of the work produced.

The discussions have been instructive from the standpoint of theory and that of physiology. It is fortunate that the essayists use this approach. However, there is need of considerably more discussions such as this, carrying the subject matter on to a presentation of pathologic aspects as well. A laryngologist's interpretation of many of the concepts presented will eventually have to be in terms of pathologic alterations of the larynx, both organic and functional, as he sees this structure in his patients. Therefore, I should like to request that the discussions be continued at another time in order to hear the opinions of the essayists on the more concrete problems the laryngologists see in the functional dysphonias and, postoperatively, in the sites of noncancerous as well as cancerous lesions.

DR. H. G. KOBRAK: I should like to ask Dr. Carhart whether the use of visible speech is practical. I had the pleasure of visiting with Dr. Steinberg in New York a few months ago. He explained and demonstrated speech patterns. Dr. Steinberg compared the study of speech patterns with the learning of shorthand. A special difficulty seemed to be that the preceding sound influences the patterns. Therefore, it seems that a considerable amount of intelligence and endurance of both teacher and student are necessary to master the subject. What are the practical results so far? What place is it expected to have in the training of the deaf?

HAROLD WESTLAKE, PH.D.: How can you recognize this glottal attack during phonation? I do not know that it has been proved that it can be detected accurately by the ear alone, although many teachers of speech believe that they can detect it, making judgments by the sharpness of the initial tone. People who do habitually speak at levels above their optimum pitches are more apt to have nodes than those who speak at their optimum pitches or below. Those who speak below their optimum pitches are more apt to have ulceration than those who speak at their optimum pitch. When one observes the manner in which the vocal folds approximate during normal phonation, the folds appear to come together relatively evenly. When observing the approximation of vocal folds in persons having vocal disorders, one often sees a notable difference in the manner of the approximation of the folds. Frequently, the vocal processes appear to come together sharply. One often notes a constriction of the supraglottic tissue.

As to the question of "neck talkers," one is apt to introduce vague terms when one is not sure of what is happening. Possibly a "neck talker" would be making unnecessary use of extrinsic muscles. Singers, and most people who have had esthetic training, unfortunately have not had a proportionate amount of scientific training. Most studies have shown an unsatisfactory appreciation of the true structural and functional picture of the mechanism.

RAYMOND CARHART, PH.D.: I should like to discuss the questions: first, that of Dr. Shapiro regarding phonetic values. The types of test to which I referred are something quite new. Speech sounds are listed in terms of carrying power, from faintest to highest. Patients often miss certain sounds because those sounds are not strong enough to be heard. In one kind of test the speech materials used are composed of the elements which carry best; with this test one measures the faintest level at which general understanding is possible. However, one can give another test in which all sounds are loud enough so that they ought to be heard with ease by the patient. One finds patients who miss some elements of speech even when they should be able to distinguish them. Such a test explores phonemic discrimination. Listeners vary a great deal in discriminatory capacity.

The second question is that of Dr. Kobrak regarding visible speech. Maybe one should look at the past. Visible speech represents a great victory for the Bell Telephone Company. Alexander Graham Bell tried to make speech visible, and failed. He was interested in devising something by which totally deaf persons could understand speech through sight without using lip reading. His hope was to bring the details of language to the person without auditory capacity. The Bell Telephone Laboratories have now invented a method which will accomplish this end. The method is rather laborious to teach to adults, but studies made with children indicate that the latter can learn it much more rapidly than adults. Furthermore, the method gives the teacher of speech a clearer idea of what the ear must analyze, and, finally, it gives a visible check on the patterns actually produced by the speech mechanism. The last is an advantage in correcting speech

patterns. Without the method one must rely on the residual hearing of the person or on kinesthetic stimulation. Consequently, one is limited by the speed with which one can teach new speech habits. With visible speech one can give the patient the picture, and he can try to produce the correct pattern.

It is too early to say what the full value of the visible speech method will be, but reports from the University of Michigan indicate that the method will be useful. Teachers of speech should remember, however, that visible speech supplements rather than supersedes the other methods of rehabilitation at their disposal.

Regular Monthly Meeting, Dec. 2, 1946

(The Scientific Meeting was presented by members of the Staff of the Illinois Eye and Ear Infirmary.)

Posterior Nasal Hemorrhage. Presented by DR. GEORGE WOODRUFF.

Under this heading will be included hemorrhages from the posterior portions of the two nasal chambers. Postoperative and traumatic epistaxis will not be discussed. The cause of the type of bleeding which I shall discuss appears to be hypertension or arteriosclerosis, or a combination of the two. In my experience it is limited to middle-aged or elderly persons and has never been encountered in a person under the age of 40. The onset is usually sudden and severe, and the bleeding is copious, though it usually ceases when the blood pressure becomes lowered, only to recur in a few hours. This sequence usually persists until moderate to severe secondary anemia occurs or until a condition of shock exists.

In treatment the first requisite is to clear the nose entirely of clots and old dark blood and to remove the fresh blood as fast as it appears, so that the site of hemorrhage may be located. Suction is useful in attaining this goal. Anesthesia and shrinkage of the nasal mucosa are provided by the use of cocaine and epinephrine.

In my experience the bleeding area is usually found in the lateral wall of the inferior meatus, back near the choanal opening, though occasionally it comes from other areas, such as the upper posterior portion of the nasal passage.

Bleeding is usually well controlled by the use of cotton tampons, 2 to 2½ inches (5 to 6 cm.) long and coated with the precipitate formed by mixing 4 per cent aqueous solution of tannic acid with 4 per cent aqueous solution of antipyrine. In a typical case such a tampon is wedged firmly into the angle between the overhang of the inferior turbinate bone and the lateral nasal wall; three more such tampons are used to fill the inferior meatus; a fifth tampon is wedged firmly between the septum and the inferior turbinate bone to aid in holding the others in position.

The greatest obstacle to success appears to be a deflected nasal septum. This often prevents accurate localization of the bleeding area and prevents proper placement of the tampons. In some cases such as this a choanal plug, pulled forward from the nasopharynx and wedged tightly into the posterior nares, may yield success. Gauze strips may then be packed against it from the anterior nares. When bleeding has not been controlled by packing, the hemorrhage has been arrested by ligation of the external carotid artery, and this should be carried out before the loss of blood becomes extreme.

When indicated, a transfusion should replace lost blood.

Routine examinations with the nasopharyngoscope have revealed the presence of a group or a plexus of blood vessels running from the posterior part of the inferior meatus downward and backward into the nasopharynx. In many older

persons these vessels are definitely dilated, and they appear to be the probable source of the hemorrhage that frequently occurs in this area. The name "naso-nasopharyngeal plexus" is suggested for this group of vessels.

DISCUSSION

DR. O. E. VAN ALYEA: While considering the subject of nasal hemorrhage aside from the area of Kisselbach, one should not lose sight of the fact that hemorrhages occur in the antrum. Some years ago an article by Hall and Thomas appeared in the ARCHIVES, in which a series of cases of hemorrhage of the antrum was reported. The theory was that sudden sneezing or violent nose blowing ruptured a vessel in the mucosal lining of the cavity. It is well, in cases of nasal hemorrhage of undetermined origin, to transilluminate or roentgenograph the sinuses, as clots in the antrum may be revealed. The authors originally treated patients for hemorrhages of this type with a Caldwell-Luc procedure; later they tried irrigation only and found that this removed the clots and stopped the bleeding. I have used this method successfully in several cases. I should like to reiterate that the antral area ought not to be overlooked as a possible source of nasal hemorrhages.

DR. E. J. BLONDER: One may often control hemorrhage in the posterior part of the nose by injecting about the bleeding area a 1 per cent solution of procaine hydrochloride or an isotonic solution of sodium chloride, using a fine needle. Blood and clots may refill the nose before a cautery can be applied. A number of years ago I started injecting procaine hydrochloride solution for control of hemorrhages of this type. After the bleeding has been controlled, the operator has an opportunity to view the ruptured blood vessel and cauterize it if he desires to do so. I seldom use the cautery, because if the bleeding is due to a sclerosed blood vessel, it may recur from the margins of the cauterized area. Powdered sugar, either inserted as a temporary pack or instilled with a powder blower, has some merit. Another excellent method is to inject a sclerosing solution about the blood vessel with a long fine needle. As a rule, I use no pack in treating these hemorrhages.

DR. MAURICE F. SNITMAN: Has Dr. Woodruff been able to determine whether the nasal bleeding is venous or arterial? Would ligation of the external carotid artery be of value when bleeding is venous in character? Personally, I do not think it would be, since venous anastomoses within the surrounding regions are of larger caliber and more abundant than the corresponding arterial supply.

DR. ROBERT HENNER: Several new products that act as hemostatic agents have been found useful in the control of severe epistaxis. I have had experience with the fibrin foam pack and the gelatin sponge. Both these materials are soaked in thrombin prior to application. The latter has more body to it for manipulation, whereas the former has pliability for insertion under the inferior turbinate bone. On one occasion in a patient with severe hypertension and profuse hemorrhage a posterior bleeder resisted all other technics such as Dr. Woodruff described. Direct application of a thrombin-soaked fibrin foam pack immediately controlled the hemorrhage. The pack is absorbed, and therefore does not require removal. Complete visualization is not necessary, but the pack must be placed in direct contact with the bleeding point.

DR. ALFRED LEWY: I have heard of an interesting experience in stopping bleeding by the use of oxidized cellulose, and I should like to hear from Dr. Woodruff on this.

DR. THOMAS C. GALLOWAY: After trying irradiation and diathermy without much success in the treatment of bleeding from hereditary telangiectasis, I have

recently been able to control this well by injecting a sclerosing solution (quinine and urethane injection U. S. P.) or "sylnasol" (a 5 per cent solution of the fatty acids of psyllium seed).

DR. GEORGE WOODRUFF: I should like to thank the discussers. So far as I know, a spontaneous hemorrhage of the antrum did not occur in any of my cases. However, it may have been overlooked, and in any event I know of no reason why it could not occur. Injections of procaine hydrochloride solution may be of value in controlling nasal hemorrhage, but in regard to these copious hemorrhages deep in the posterior portions of the nose I am somewhat doubtful of their value. Theoretically, it would be a good thing to have suture material tied to the tampons and fastened on the face. However, it complicates the packing, and with the technic used, none have so far slipped backward.

Ligation of the external carotid artery has been successful in all 4 cases in which it has been used. Perhaps this is an argument that the hemorrhages were all arterial, but I am not sure. I have had no experience with ligation of the internal maxillary artery, but it seems that it would be a more difficult procedure than ligation of the external carotid artery.

Nasal Melanoma. Presented by DR. ALFRED LEWY.

This man, a Negro aged 44, first came to the Illinois Eye and Ear Infirmary in October 1944. A dark bluish tumor filled the left side of the nose. The roentgenogram showed the left maxillary sinus to be cloudy, owing to thickening; the other sinuses appeared normal. A biopsy was made, and the pathologist's report stated definitely that the tumor was melanoma. It appeared to be about the consistency of an ordinary nasal polyp and was apparently attached to the septum. All that part of the septum including the base of the tumor was removed by cautery knife—practically the entire cartilage of the septum and perhaps some of the bone; I do not know just how far the removal went, because the operation was performed with the electrocautery knife.

After a few months there was recurrence, the tumor being almost as large as the original one. The incision was widened, and this time there was an invaded area at the junction of the septum and the ridge of the nose; all of this invaded tissue was removed. Subsequently there were three recurrences, each under the ridge of the nose where the septum would have joined had there been any septum left. Each time the growth was destroyed by the electric needle. It has now been several months since any trace of the melanoma was found.

Melanoma is exceedingly rare in this area and is especially rare in Negroes. McCullen says it is a curious fact that among horses melanoma practically never occurs in any but white or gray horses and that in every case in which melanoma was observed at autopsy it was in a white person.

The patient returns at frequent intervals for observation. Nothing has been found but a small depression of the ridge of the nose. There is a spot on the forehead which resembles melanoma, but the patient says it has been there for as long as he can remember. This has been left untouched.

An Unusual Laryngeal Tumor in an Aged Man. Presented by DR. ALFRED LEWY.

J. N. V., a man aged 81, entered the Illinois Eye and Ear Infirmary on Feb. 16, 1945, complaining of hoarseness and respiratory obstruction. A round whitish neoplasm, smooth and shining, was seen occupying the introitus of the larynx so that nothing could be seen below it. With the region under local anesthesia, the

growth was easily divulsed by direct laryngoscopy, leaving a smooth raw surface on the left side of the larynx, above the cords. The patient left the hospital on February 19 in good condition. The tumor was round, had a smooth, shining capsule and was clinically consistent with fibroma, a judgment which was confirmed by the first histologic report.

The patient returned on July 19 with a recurrent growth obstructing the larynx. It appeared as an irregular grayish mass. It was removed, after induction of local anesthesia, by laryngofissure, and a median tracheotomy was done. Sections from this tumor were studied by several well known pathologists, including two visitors from England, and the tumor diagnosed as fibrosarcoma.

In spite of his age, the patient showed no ill effects and left the hospital within a few days. He was sent to the Illinois Research Hospital for radiation therapy, but returned to the Illinois Eye and Ear Infirmary at intervals for observation. The last time he was seen before his death he had removed the tracheal tube, the tracheostoma had nearly closed, and there was no evidence of recurrence; so another tracheotomy was not performed.

The second tumor measured 3 by 13 cm. It was attached at the original site and hung down into the trachea. It is remarkable that the patient could breathe as well as he did.

On Jan. 3, 1946, the patient was brought to the hospital and died before another tube could be inserted. The interesting features in this case are the age of the patient, the size of the tumor, the mistaken original diagnosis and the mistaken judgment of permitting the tracheotomy to close.

DISCUSSION

DR. MAURICE F. SNITMAN: There are a number of cases of fibroma in which the determination of a sarcomatous quality is difficult. Furthermore, in the same specimen some areas are definitely more cancerous than others; accordingly, further sections of the biopsy specimen may be of assistance in establishing a more accurate diagnosis. Because of the poor response of fibrosarcoma to irradiation, surgical excision is usually the proper method of treatment. It would be inadvisable to irradiate the site postoperatively. Such treatment might prevent further surgical attention to a local recurrence.

DR. ALFRED LEWY: The biopsy specimen of the first tumor was studied extensively. After a long search, two pathologists stated that some cancer cells were found in the outlying area. If they were there, they were overlooked at first.

Ear, Nose and Throat Survey of Children in the School for the Deaf at Jacksonville, Ill. Presented by M. TAMARI and R. SPEAS.

In a survey suggested by the Illinois Eye and Ear Infirmary and with the cooperation of the State Department of Public Welfare in Springfield, Ill., 398 students ranging in age from 4 to 21 years were examined otologically. The main purpose was to classify the type of deafness and to determine the extent of residual hearing. In collaboration with the teaching staff it was possible to discuss individual treatment and future educational training. Anatomic classifications were illustrated in histologic sections and clinical types of classification recommended accordingly.

The survey was accomplished on a special form which could be used as a basis for future studies. It was adapted, with some changes, from similar forms used in rehabilitation centers of the Army and the Navy.

Of the entire group, 289 (72 per cent) were congenitally deaf, 46 (11.6 per cent) had postmeningeal deafness and 63 (15.8 per cent) became deaf after chronic aural infections. Thirty-eight (13 per cent) of the congenitally deaf children had no vestibular reactions; 16 (32 per cent) of those with postmeningeal loss of hearing had positive labyrinthic responses.

In evaluation of the present status of the examination and school training of deaf pupils it was found that there is a period of advancement. The progress is indicated by the policy of the school to admit only children in whose cases a diagnosis of the type of deafness has been established by complete otolaryngologic examination. From the otologic point of view there is still a great necessity for intensive study of the genetics of deafness, as well as an urgent duty to inquire into the marital status and familiar relationship more carefully. Family histories are started in the school, and forms for statistics and pedigrees are planned. Beginning with this survey, efforts have been made in collaboration with the teaching staff to establish a precise diagnosis of every type of deafness. The clinical classification of deafness was found helpful in the organization of individual training.

It was learned that deafness acquired through meningitis causes such symptoms as noises and dizziness, not present in the congenitally deaf. It was interesting to note that a considerable number of the subjects whose hearing loss was postmeningeal had normal labyrinthic responses.

It was recommended that children with residual hearing should be watched carefully for nasopharyngeal changes after contraction of colds. Repeated salpingitis may reduce and damage hearing islands. Tonsillectomy and adenoidectomy should be performed more frequently. Residual lymphatic tissue or recurrent hypertrophy should be treated by repeated operation, and excessive lymphoid tissue should be treated with radon or roentgen radiation. Purulent ears should be treated conservatively for only a limited time; then the patients should be referred to an otologic department. Children with postmeningeal deafness should be placed in occupations in which excessive movements of the head can be avoided. Intensive tinnitus should be classified as to origin and treated accordingly.

Large schools for the deaf should establish an otologic service, with all facilities for examination, treatment and the fitting of hearing aids at the school. This would not only help to establish a precise diagnosis and the extent of deafness but also prevent loss of residual hearing.

DISCUSSION

DR. SHERMAN SHAPIRO: I want to say a few words about the diagnostic problem in these cases. Although I happen to be a "tuning fork man," I believe that in a study of this type the audiometer will be of more value than tuning forks; particularly, a sweep range type of machine would be invaluable for discerning islands of hearing. Tuning forks can be used also, but only according to a standardized technic such as the one advocated by the Committee on Tuning Forks, appointed by the Academy of Ophthalmology and Otolaryngology a number of years ago.

Directory of Otolaryngologic Societies *

INTERNATIONAL

First Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit, Mich.
Place: Chicago. Time: June 21-25, 1948.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
President-Elect: Dr. C. H. McCaskey, 20 N. Meridian St., Indianapolis, Ind.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 12-17, 1947.

American Broncho-Esophagological Association

President: Dr. Millard F. Arbuckle, 539 N. Grand Blvd., St. Louis.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.

American Laryngological Association

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.
Place: Old Homestead, Va. Time: April 14-15, 1948.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lyman G. Richards, 15 Whiting Rd., Wellesley Hills, Mass.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Chalfonte-Haddon Hall, Atlantic City. Time: April 7-9, 1948.

SECTIONS:

Eastern.—Chairman: Dr. Robert F. Ridpath, 18th and Chestnut Sts., Philadelphia.
Place: New York. Time: Jan. 16, 1948.
Southern.—Chairman: Dr. Walter T. Hotchkiss, 541 Lincoln Rd., Miami Beach, Fla.
Place: New Orleans. Time: Jan. 23, 1948.
Middle.—Chairman: Dr. Oliver E. Van Alyea, 135 S. La Salle St., Chicago.
Place: Columbus, Ohio. Time: Jan. 19, 1948.
Western.—Chairman: Dr. Colby Hall, 1136 W. 6th St., Los Angeles, Calif.
Place: San Francisco. Time: Jan. 31-Feb. 1, 1948.

American Otological Society

President: Dr. Bernard J. McMahon, 634 N. Grand Blvd., St. Louis.
Secretary: Dr. Gordon D. Hoople, 713 E. Genesee St., Syracuse, N. Y.
Place: Old Homestead, Va. Time: April 12-13, 1948.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Alfred Schattner, 115 E. 61st St., New York.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven, Conn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.